

PORTAL HYPERTENSION

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JACKSONIAN PRIZE ESSAY
ROYAL COLLEGE OF SURGEONS 1956

A CONTRIBUTION TO THE STUDY OF
**PORTAL
HYPERTENSION**

BY

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PREFACE

THE following contribution to the study of Portal Hypertension was submitted for the Jacksonian Prize of the Royal College of Surgeons for the year 1956. Its format was dictated by the requirements of that ancient prize which stipulate that the essay shall not exceed 75,000 words in length. No attempt has, therefore, been made to summarise the vast amount of scientific work which has recently been published on the hepatic circulation and on liver disease, although I have attempted to relate my clinical experience to the scientific findings.

I am fully conscious of the debt that I owe to the many physicians and surgeons who referred patients, and to my colleagues and assistants. To all I wish to record my most sincere thanks for their help. Individual mention must be made of those who have referred the majority and given me special encouragement. Mr Lawrence Abel, Dr G. P. Baker, Dr Kenneth Black, Dr Ronald Bodley Scott (who referred Case 1, among many others, and provided the initial stimulus), Dr Geoffrey Bourne, Sir Stanford Cade, Dr J. Caplan, Dr Edward Cullinan, Sir Daniel Davies, Dr H. K. Goadby, Sir Neil Hamilton Fairley, Dr Alfred Franklin, Dr John Harman, Dr Philip Harvey, Mr Basil Hume, Dr John Hunt, Dr Kenneth Robertson, Mr Norman Tanner, Dr P. G. Todd and Professor A. W. Woodruff.

Dr Ronald Bowen has given practically all the anaesthetics with his usual skill which has always given me great confidence, even during the most difficult operations.

The development of an efficient method of obtaining portal venograms has been of vital importance and depended very largely on the excellent help given to me by Dr R. A. Kemp Harper and on the unstinted co-operation of Dr George du Boulay and Dr Benjamin Green of St Bartholomew's Hospital and Dr J. J. Stevenson at the Royal Marsden Hospital.

Professor W. V. Mayneord and Dr E. H. Belcher of the Royal Marsden Hospital have enabled me to estimate the speed of flow in the portal vein and to Dr Belcher I would like to express my particular thanks for the number of hours that he has waited in the operating theatre in order to be able to estimate the speed of flow after the construction of a venous anastomosis. Mr H. C. Hodt has shown his expected ingenuity in constructing the double scintillation detector. Dr R. A. Allen of the Atomic Energy Research Establishment at Harwell has estimated for me the content of arsenic and gold in certain cases of cirrhosis hepatis by the fascinating method of irradiation spectrometry.

PREFACE

Dr Hermann Lehmann, at the suggestion of Professor Bruno Mendel, has for five years worked on the assessment of liver function by estimating the pseudocholinesterase content of the serum, information which I have found of great value, and Professor J W S Blacklock and the Research Committee of St Bartholomew's Hospital have enabled this work to be done with the aid of a grant from the Medical Research Council

Mr I Cull and Miss J Akister have prepared tables and illustrations for reproduction I am especially grateful to Mr N K Harrison and his staff in the Photographic Department of St Bartholomew's Hospital and to Miss J Hunt of the Royal Marsden for providing me with the photographic illustrations

Miss E C Hall, Miss J Lightfoot and Miss M Rutherford of St Bartholomew's Hospital have supervised the nursing of the majority of the patients with incomparable skill and kindness The following sisters of St Bartholomew's Hospital, Miss B Bartlett, Miss J Christie, Miss V Jenkins, Miss A Mallet, Miss M Rutherford and Miss J Souttar, and Miss G Berdach of the Royal Marsden Hospital, have organised the operating theatres and taken the cases in a way which could not have been bettered My theatre orderly, Mr Henry Dossett, assisted in devising the hornpipe position which I have found most useful

Lastly, I would like to express my thanks to Miss Bernice Cattle and Miss Audrey Harby for keeping the records with such care and for helping to prepare this book for publication, and my appreciation of the great consideration shown to me by Mr Charles Macmillan of E & S Livingstone Ltd

ALAN H HUNT

London, 1957

ACKNOWLEDGMENTS

A number of the illustrations have been reproduced before sometimes in more than one journal in order to demonstrate different aspects of the subject of portal hypertension. For example Figure 86 was originally reproduced by Dr du Boulay and Dr Green to show the position of the cassette tunnel on the operating table and then used again by Mr Lawrance Mr Whiteley and myself to show the hornpipe position. I wish to express my most sincere thanks to Dr L. W. Proger of the Royal College of Surgeons for supplying me with Figure 98 and to the editors of the various journals and books concerned for their permission to reproduce these illustrations.

Proceedings of the Staff Meetings of the Mayo Clinic and Drs Douglas Baggenstoss and Hollinshead for Fig. 1

British Journal of Clinical Practice for Figs 2 3 15 16 31A 38A 48

American Journal of Roentgenology for Figs 7 10 22A and B

British Journal of Radiology for Fig. 9

Lancet for Figs 14 20 86

Proceedings of the Royal Society of Medicine for Figs 17 53 112

British Medical Journal for Figs 37 66

Messrs J. & A. Churchill for Fig. 40

Masson et Cie Paris for Figs 49B 89 92

Journal de l'anatomie et physiologie for Fig. 52

Appleton Century Crofts New York for Figs 81 97

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CHAPTER 1

INTRODUCTION

THE portal vein exists to carry blood from the stomach, intestines, spleen, and pancreas to the liver. An obstruction to this flow is necessarily unphysiological whatever it may be. It leads to stagnation of portal blood, a rise of portal venous pressure, and congestion of the organs which drain into the portal tree, particularly the spleen. The body's reaction is to attempt to outflank the obstruction by diverting portal blood into the systemic circulation through communicating vessels. In the cardiac end of the stomach and in the oesophagus these develop into a mass of varicose channels from which devastating haemorrhages are liable to occur. The relief mechanism suddenly thus becomes a serious threat to life.

This, briefly stated, is the condition that has come to be known as portal hypertension. It is not a disease but a state, most commonly caused by a cirrhotic liver. The surgeon is becoming interested in operations which can relieve or palliate this state, and if in so doing he can succeed in improving upon nature by diverting all the portal blood directly into the systemic circulation it is necessary to find out what effect such apparently unnatural measures have upon the body.

The circulatory state of the portal tree can become so altered that accepted physiological conceptions may have to be modified or discarded in considering the problem. It is necessary for new ideas to be subjected 'to fair trial lest they be dissipated in an atmosphere of tradition and authority' (of Wangensteen). The present report, which analyses 250 personal cases of portal hypertension, has therefore been written with the purpose of examining the new findings and assessing the new methods of treatment. I hope it will help to clarify certain issues on which there is at present no general agreement. I intend that it shall be a statement and demonstration of what has been encountered and what has resulted from treatment, unencumbered by speculative theorems.

The patients were in no way selected. They constitute a consecutive personal series, but there has, of course, been a sifting of material in the mode of reference. For example, cirrhotic patients who had responded favourably to medical treatment alone were not often referred because there was no need for a surgical opinion in this group. It was the more complicated cases with haemorrhage, ascites, or jaundice that were sent for investigation or treatment. Many were desperately ill, some in the very terminal stages of their disease, and they had been sent in the hope that surgery might possibly have something new to offer. None has been omitted from the

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analysis The follow up has been complete, thanks to the co operation of the surviving patients themselves and their doctors, physicians and surgeons. The first patient was seen early in 1947 and the last to be included in the series in June 1956, so that the results have been assessed on an appreciable follow up, in some cases of many years and never less than six months. Relevant material from patients seen during the latter part of 1956 has also been included in the discussion without in any way prejudicing the analysis.

‘When animal existence is supported by any other than the usual admirably contrived means, it cannot fail to excite the attention of the philosopher, since it shows to him the powers and resources of nature’—ABERNETHY, 1789

CHAPTER II

ANATOMICAL CONSIDERATIONS

THE PORTAL VEIN

KNOWLEDGE of the normal anatomy and of the variants of the portal vein and its tributaries is essential to the proper understanding of portal obstruction, particularly of the extrahepatic type. Excellent recent studies have been published by Gilfillan, Douglass, Baggenstoss, and Hollinshead, Falconer and Griffiths; and Child. The accompanying diagram of the normal anatomy (Fig. 1) is generally accepted as the usual arrangement. All workers

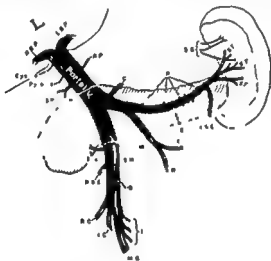


FIG. 1
The portal vein and its tributaries
From Jones to Baker et al. & Hollinshead

emphasise the great variations that may be encountered within the 'normal' range and how these variations may affect the development and treatment of the abnormal state. As examples, the left gastric, or coronary, vein may enter splenic or portal and its position may have a significant bearing on the development of oesophageal varices. radicles from gallbladder, duodenum, pancreas and stomach may or may not enter the trunk of the portal vein itself. The position, length diameter tortuosity, and lie of the portal vein vary from case to case (Rousselot, 1953). Abernethy in 1789 even described a portal vein opening directly into the inferior vena cava in a case of trans

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position of viscera Duplication and anterior placement of the portal vein have also been described

The operating surgeon must be able to translate academic anatomical knowledge into a demonstration of projection anatomy so that he may know



FIG 2
Normal splenic venogram Thrombocytopenic purpura Portal venous pressure 80 mm water Diffuse distribution of blood throughout liver

the exact state of the portal vein in each case To him only the anatomy of the individual is important, and he should know it if possible before beginning the dissection Portal and splenic venography are the radiographic methods employed, prior to palpation and dissection The accompanying figures (2, 3) are two examples of unimpeded normal portal circulation and provide the standard by which subsequent illustrations may be judged The

ANATOMICAL CONSIDERATIONS

blood flows directly and without diversion from splenic or superior mesenteric through the portal vein into the liver

Definition of the portal vein is not necessarily complete by either method owing to displacement of blood containing the radio-opaque diiodone by blood from another part of the portal tree. This normal 'streamlining' or laminar flow (Helps and McDonald 1954 Dreyer 1954) is well shown in Fig 3. The speed of flow about 9 cm per second is too slow for there to be more than partial mixture so the splenic blood stream is either pushed aside, split or rotated by the blood coming from the superior mesenteric vein. Laminar

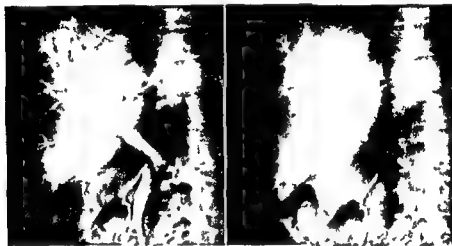


FIG 3

Portal venogram in a case of gallstones. To show the normal liver pattern and a rapid though laminated or streamlined flow through the portal vein. Speed of flow 80 cm per second. Portal venous pressure 55 mm water.

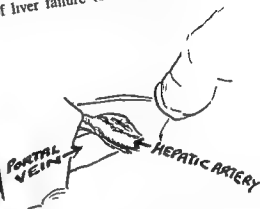
flow can easily be confused with thrombosis but the composite picture obtained by both methods and by serial radiographs gives accurate information. Within the liver the blood is usually evenly distributed and selective flow from one group of portal radicles to any particular segment of the liver has not been demonstrated. Raven came to the same conclusion using slightly different methods.

THE HEPATIC ARTERY

The hepatic artery normally arises from the coeliac axis and divides into two branches which lie in front of the corresponding branches of the portal vein as they pass up to the liver. The unusual must again be anticipated. An abnormality which may seriously interfere with the dissection of the portal

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vein has been encountered four times. The hepatic artery itself, or a large branch, has been found hooking round the right border of the vein (Fig 4). On one occasion the operation had to be abandoned. On another it made the placing of the anastomosis faulty so that the portal vein was kinked over the artery, the shunt became thrombosed and the patient died (Case No 108). On the third it was just possible to separate hepatic artery and portal vein completely so that an excellent and uninterrupted shunt could be constructed. Unfortunately the man died post-operatively of liver failure (Case No 245).



B

FIG 4

Patient No 108. Abnormally placed hepatic artery which can be seen (arrowed) hooking round the portal vein on its posterior right and then anterior aspects. This and the proximity of the pancreas rendered porta caval anastomosis impossible.

At postmortem the shunt was found to be satisfactory. The artery was the common hepatic, intact and undamaged, which arose from the superior mesenteric (an anomaly which has been described by Henle 1872, and by Browne 1940). The fourth patient was similar, but he fortunately made an excellent recovery from his porta-caval anastomosis and will, I hope, give us no opportunity for further dissections.

Other anomalous origins (see Henle, 1872) are encountered from time to time such as that of the left hepatic artery arising from the left gastric, which becomes of special importance in considering the operation of hepatic arterial ligation. The purpose of this operation is to cut off the whole arterial supply to the liver, and this will mean an extensive search to be sure that no major vessel is missed.

ANATOMICAL CONSIDERATIONS

THE INFERIOR VENA CAVA

The successful construction of a direct porta-caval anastomosis depends to some extent on clean dissection of the vena cava right up to and even behind the caudate lobe of the liver. At and just below this point there are two, three, or even four veins draining into the vena cava from the liver itself, the capsule of liver, or other surrounding structures. If they are torn they cause troublesome bleeding, if they are recognised they can easily and bloodlessly be divided between ligatures.

On one occasion the inferior vena cava was found to be double. It compelled a wider dissection but did not interfere with the construction of an anastomosis.

THE RENAL ARTERY AND VEIN

The single renal artery usually lies behind the vein and allows the successful completion of an end to side lieno-renal anastomosis. The artery, however, is often multiple (from the segmental origin of the organ) and important branches may lie in front of the renal vein, sometimes making smooth coaptation with the splenic impossible. If they are small they can occasionally be sacrificed, but only after trial clamping for about ten minutes with a Blalock clamp. If they are large it is better to sacrifice the kidney, strip the vein of its enclosing arteries, and make an end-to-end anastomosis with the splenic vein.

Anomalies in the renal vein, though much less common than those of the artery, are sometimes found and can be circumvented with a little ingenuity (Case No. 219).

CHAPTER III

THE CAUSES OF EXTRA AND INTRAHEPATIC PORTAL OBSTRUCTION

GENERAL CONSIDERATIONS

ANATOMISTS have been aware for more than 120 years of the vascular communications that exist between the portal and systemic venous circulations and they have, so far as the restricted scope of the dissection of a few cadavers allowed, recognised a condition similar to what we now term portal hypertension. The establishment during the present century of special centres for the study and treatment of difficult diseases has enabled physicians, surgeons, pathologists, physiologists, anatomists, and radiologists to co-operate in elucidating facts on an altogether more expansive scale. Allen O. Whipple's Spleen Clinic at the Presbyterian Hospital, New York, is such a centre, and from there have originated many of the current ideas on portal hypertension. Rousselot, for example, measured the pressure in the splenic vein on the operating table and was able to define, in 1936 and 1940, the causes of extrahepatic portal obstruction. Out of this grew the classification (Whipple, 1945) that has served well during the short period of ten years that has been available for most of us in this country for the scientific study of 'civilian' surgery.

There are two main types of obstruction—the extrahepatic, in which the block is beyond the liver and interferes with the flow of blood to that organ (which is normal in other respects), and the intrahepatic, in which the fibrosis of the liver itself constitutes the obstruction. In addition, portal hypertension can be caused by an interference in the return of blood from the liver to the heart, as in obstructions to the hepatic veins and the thoracic part of the inferior vena cava (the Budd Chiari syndrome) and in constrictive pericarditis.

How was it that the early anatomical studies did not lead sooner to the classification propounded by Whipple? One explanation is that the subject is of such complexity involving so many different organs in a jumble of striking and inconstant symptoms and signs that a false theory put clinicians and pathologists on to a wrong idea. In 1894 Guido Banti of Florence, deceived by the enormous spleen which made itself evident as the initial abnormality in certain cases of progressive cirrhosis hepatis, described a disease which was named after him. The spleen, according to Banti, was primarily at fault and the infection whatever it was, spread from spleen to liver and gave rise to a disease which 'progresses slowly but it inexorably

CAUSES OF OBSTRUCTION

drags the patient to death' Banti had, as it were, reversed the charges. His writings were forceful and served a useful purpose in transferring interest from the dissecting room and postmortem department to the wards. But his ideas died hard. In my first pathological notebook, compiled in 1931 under the instruction of the late Dr A. G. Gibson of Oxford, I have a sketch of a typical Banti spleen showing a 'mycelium' which, we were led to believe, had spread to the liver and ultimately killed the patient. A different method of staining shows the 'mycelium' to be iron. Work on the problem of splenic anaemia led to the distinction between congestive splenomegaly and the primary type of this disease in which there was no obstruction to the flow of portal blood. Dock and Warthin in 1904 considered that the splenic condition could be secondary to an obstruction in the portal vein. An authoritative statement on the subject was written by Eppinger in 1920 and it was during the 'twenties that most workers finally discarded Banti's theory. The eponym lingered as 'Banti's syndrome,' a condition in which splenomegaly is associated with oesophageal varices, and it is right that the name of an original enthusiast should be perpetuated, even though his theory was proved to be wrong.

Already in 1901, however, Langdon Brown writes concerning pylethrombosis that haematemesis and melaena are accepted symptoms of the associated portal stasis and occurred together in twenty-two out of forty-one patients. His article proves that the condition caused by portal obstruction was well understood at that time but that much of this understanding appears to have been lost or ignored. McIndoe's beautiful study appeared in 1928, in which he demonstrated the distorted vascular pattern in cirrhosis and undoubtedly understood the full implications of portal obstruction and elevated portal pressure.

CHAPTER IV

EXTRAHEPATIC PORTAL OBSTRUCTION

THE different types of extrahepatic obstruction encountered in the present study are set out in the table (Fig 5). They number only thirty two (or 12.8 per cent) of the 250 patients. Comparable proportions are given by other workers e.g. Linton and Ellis (1956) twenty five of 131 patients

EXTRAHEPATIC OBSTRUCTION Total 32 Cases (12.8% of all)

CONGENITAL OBLITERATION Total 19 cases

11 Females, average age 11 years 8 Males, average age 15 years

- 13 Cavernomatous replacement of Portal vein
- 2 Stricture of Portal vein
- 2 Cruveilhier Baumgarten anomaly
- 1 proved Suppurative Ophthalmitis
- 1 presumed Congenital

THROMBOSIS OF PORTAL VEIN Total 7 cases

- 2 Following abdominal Trauma,
- 3 Infective two following Appendic abscess
- 2 Associated with blood disorders
Lymphosarcoma Myeloid Leukaemia

COMPRESSION OF PORTAL VEIN Total 3 cases

- by Hypernephroma right Kidney
- Displaced left Kidney
- Primary Carcinoma of Liver

INVASION OF PORTAL VEIN 2 cases

In both by Carcinoma of the Stomach

SPLenic ARTERIO-VEINous ANEURYSM 1 case

FIG 5

CONGENITAL PORTAL VEIN OBLITERATION

There were nineteen in this group of which one was diagnosed on presumptive evidence only

Thirteen showed cavernomatous replacement of the portal vein a condition well recognised since the early years of this century especially in Germany Beitzke in 1910 and Emmerich in 1912 give accurate descriptions

EXTRAHEPATIC PORTAL OBSTRUCTION

of the condition and Klemperer in 1928 related it as a causative factor to Banti's disease

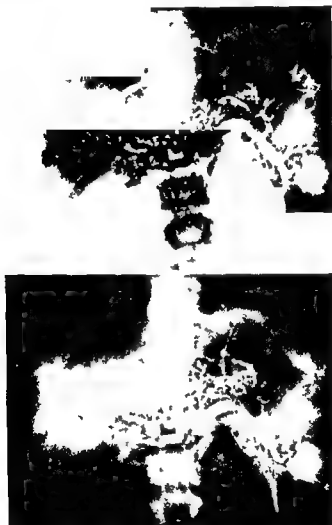


FIG 6

Case No 219 Double splenic venogram showing congenital portal vein obliteration with cavernomatous replacement. Anastomotic channels in diaphragm well shown. Splenic vein present

Figs 6 7 8 and 9 illustrate the condition in its many bizarre forms and provide a striking contrast to Figs 2 and 3. In none does the portal vein exist. It has been replaced by or transformed into (as many writers prefer to

PORTAL HYPERTENSION

describe it) a mass of cavernomatous vessels They may even form a vascular 'tumour' enveloping the common bile duct in the free border of the lesser omentum (Fig 18, p 22) They transport blood to the liver as shown by Pick in 1909 who called them the 'hepatopetale Kollateralbahnen' which he demonstrated in a case of total haemangiomatous portal vein obliteration



FIG 7
Case No 90 Cavernomatous replacement of portal vein in a boy of 2
Portal venogram via a left omental vein

Fig 11 shows how both X ray methods are sometimes necessary for a complete demonstration of such an obstruction and that no major channel exists in the portal tree In some cases the cavernoma is composed of small channels in some large but in every case the veins are thin walled perhaps with thickened patches tortuous baggy and difficult to handle It is well nigh impossible to dissect up a length of such a vein It disintegrates into a beaded thread useless for the purpose of vascular anastomosis Figs 11c 18a show the appearances that cavernomas may present at operation The

EXTRAHEPATIC PORTAL OBSTRUCTION

vascular transformation is not superficially different from any other cavernomatous replacement

Theories of how the portal vein becomes occluded and replaced have been numerous, but for the group under consideration it seems that an extension of the process that normally brings about an obliteration of the umbilical



FIG. 8

Case No 119. Cavernomatous replacement of portal vein with marked gastro-oesophageal varices (Also mildly cirrhotic presumably arsenical in origin) Female aged 37

vein and the ductus venosus at birth is the most likely (Thompson, 1940). The diagram (Fig. 12) shows where the umbilical vein and ductus venosus link up with the left branch of the portal and right hepatic veins. In the Cruveilhier Baumgarten disease (two examples of which have been encountered in this group) the obliterative process obstructing the ductus venosus is supposed to extend to involve the hepatic part of the bifurcation of the portal vein without obliterating the umbilical. The findings in cases such as that



FIG 9

Case No 85 Extrahepatic portal obstruction with cavernomatous replacement of the portal and superior mesenteric veins Inferior mesenteric vein large

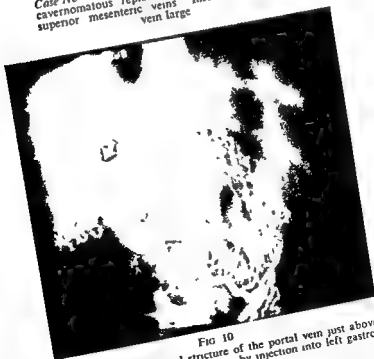


FIG 10

Case No 91 Congenital stricture of the portal vein just above entrance of cystic veins Venogram by injection into left gastro-epiploic vein

EXTRAHEPATIC PORTAL OBSTRUCTION



FIG II

Case No. 251 (not included in the table Fig 5) Above (A) portal and (B) splenic venograms in a case of splenic and portal vein obliteration by carcinoma of the pancreas. The portal vein is indistinctly shown beyond the site of obstruction (arrowed). Below (C) operation photograph of the same turgid veins filling gastrohepatic omentum.

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illustrated in Fig 13A provide strong support for this theory (see also Armstrong *et al* 1942, Jahnke *et al* 1954) The portal blood reaches a blind end in the porta hepatis before being diverted to the anterior abdominal wall although a little may percolate out into the liver

There have also been two cases of portal stricture (Fig 10) identical with the case described by Mahoney and Hogg It can be seen on the venogram that the obstruction is to the trunk of the portal vein itself and that the umbilical vein is not patent Further extension of the obliteration downwards would blend with the picture of cavernomatous replacement

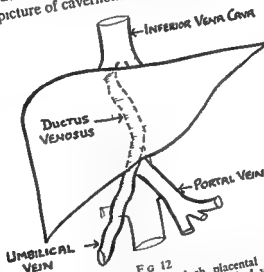


Diagram to show route by which placental blood reaches the inferior vena cava The hatched vessels the umbilical vein and the ductus venosus are normally obliterated at birth

In all these patients with congenital obstruction there was no history whatsoever of any event or illness to which could be attributed the portal obliteration with the exception of the nineteenth a boy of 6 who had had neonatal suppurative omphalitis with an epigastric abscess His case is borderline between what may be considered as congenital and acquired At operation however no trace of the portal vein could be felt and his cavernoma was characteristic of the congenital type He is therefore considered as such In all except Case No 119 the liver was normal either on examination at operation or by microscopical examination

Sex and Age Incidence

There were eleven females average age 19 and eight males average age 15 Six were less than 10 years of age when first seen six were between 10 and 20 three between 20 and 30 and four between 30 and 40 Some had had

EXTRAHEPATIC PORTAL OBSTRUCTION



FIG 13

Case No. 242. A Splenic venogram to show Cruveilhier Baumgarten disease. B Portal venogram to show portal (and splenic) vein thrombosis three weeks after lino-renal anastomosis (see Fig. 75).

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illustrated in Fig 13A provide strong support for this theory (see also Armstrong *et al*, 1942. Jahnke *et al*, 1954) The portal blood reaches a blind end in the porta hepatis before being diverted to the anterior abdominal wall, although a little may percolate out into the liver

There have also been two cases of portal stricture (Fig 10) identical with the case described by Mahoney and Hogg It can be seen on the venogram that the obstruction is to the trunk of the portal vein itself and that the umbilical vein is not patent Further extension of the obliteration downwards would blend with the picture of cavernomatous replacement

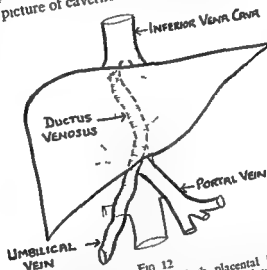
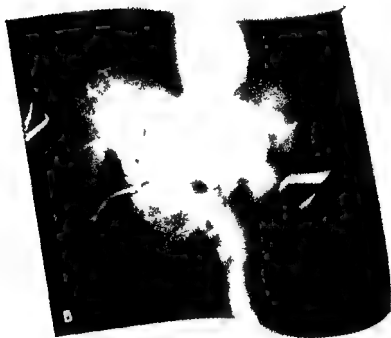


FIG 12
Diagram to show route by which placental blood reaches the inferior vena cava. The hatched vessels the umbilical vein and the ductus venosus are normally obliterated at birth

In all these patients with congenital obstruction there was no history whatsoever of any event or illness to which could be attributed the portal obliteration with the exception of the nineteenth, a boy of 6 who had had neonatal suppurative omphthalitis with an epigastric abscess His case is borderline between what may be considered as congenital and acquired At operation however no trace of the portal vein could be felt and his 'cavernoma' was characteristic of the congenital type He is therefore, considered as such In all except Case No 119 the liver was normal either on examination at operation or by microscopical examination

Sex and Age Incidence.

There were eleven females average age 19 and eight males average age 15 Six were less than 10 years of age when first seen six were between 10 and 20 three between 20 and 30 and four between 30 and 40 Some had had



Case No 242 A Splenic vein
venogram to show portal (and
splenic) anastomosis

Case No 242
A Splenic vein
venogram to show portal (and
splenic) anastomosis

PORTAL HYPERTENSION

chronic anaemia for many years and three of the adult cases had had their spleens removed in childhood. The age of these patients does not, therefore



FIG. 14

Case No. 66 Localised thrombosis of the portal vein best shown in the venogram in front of the first lumbar vertebra, where it occupied most of the lumen as shown in the second photograph

indicate the age at which their symptoms began. The average age is considerably less than in cirrhosis

EXTRAHEPATIC PORTAL OBSTRUCTION

The two with Cruveilhier Baumgarten disease, in whom the umbilical vein served as a channel for partial portal decompression and in whom the evidence of a congenital origin for their disease is strongest, were both adults, though they both had a long story of ill health

DISTINCTION BETWEEN CONGENITAL AND ACQUIRED OBLITERATION

It has been questioned whether it is justifiable to distinguish between the congenital and acquired extrahepatic occlusion. Gibson and Richards, for example, do not consider that any of six cases they studied were congenital



FIG. 15

Case No. 183 Double portal venogram showing portal vein obliteration probably the result of abdominal trauma with cavernomatus replacement. The spleen has been removed. Much blood is being diverted up oesophageal varices.

in origin and that thrombosis is therefore the cause. When, however, there is no antecedent history of an illness and where there is either no residual trace of the portal vein or when there is some well localised constriction without any other abnormality (as in Figs 10 and 13A), it is difficult to believe that a pathologically acquired thrombosis is the cause. If, however, a thrombosed portal vein is palpable and its recanalised remnants can be seen in venography (Figs 13B and 14), the condition may then be considered as acquired. These are the criteria on which the present cases have been classified. Clinically and at operation, however, it is sometimes very difficult to differentiate between the two types (Fig 15) and certain cases must always remain borderline.

ACQUIRED OBLITERATION OF THE PORTAL VEIN

The portal vein was adjudged to have been thrombosed in seven patients compressed in three and invaded by carcinoma in two



FIG 16

Case No 157 Double solenic venogram showing portal vein obliteration following portal pyephebitis due to appendix abscess
Liver normal

THROMBOSIS OF THE PORTAL VEIN

Two otherwise healthy individuals related the onset of their symptoms to *abdominal trauma* one a street accident and the other a deep injury to the right side playing rugby football (Fig 15) Heller Kiel (1904) and Rousselot (1936) describe somewhat similar cases

EXTRAHEPATIC PORTAL OBSTRUCTION

In three patients the symptoms of portal obliteration followed *abdominal infection* in two an appendix abscess, and in the third there was a story of



FIG 17

Infective Cholangitis

Case No. 53 Double splenic venogram showing almost complete portal vein obliteration in a case of biliary cirrhosis due to common hepatic duct obstruction. Extreme stasis. Diversion of blood up gastro-oesophageal varices.

an acute infective disease with ascites many years previously (see Langdon Brown, 1901, and Simonds, 1936). The appearances in the third case were astonishing (Fig. 18). At operation the portal vein felt like a hard rod firmly

PORTAL HYPERTENSION



FIG III

Case No. 248. Splenic venogram and operation photograph of cavernomatous replacement of thrombosed portal vein (infection). The cavernoma lies above the distended gallbladder and envelopes the common bile duct. The tortuous vessels are almost the size of a finger.

EXTRAHEPATIC PORTAL OBSTRUCTION

embedded within the vascular mass and inseparable from it or the bile duct. It lay between the great cavernomatous channels above and the inferior vena cava below, barring direct anastomosis between the two. The cavernomatous vessels were nearly as big as an index finger. (Figs 17 and 20 are also



FIG. III

Case No. 104. Portal venogram showing blood travelling to the liver up biliary veins alongside the portal vein which was fully patent. Dilatation of these collateral channels would lead to the formation of a cavernoma.

illustrative of portal thrombosis due to infections but in both cases the initial obstruction was due to cirrhosis of the liver.)

There was splenic vein thrombosis in a case of *lymphosarcoma* and total thrombosis of the portal tree with cavernomatous replacement in a case of *myeloid leukaemia*.

CAVERNOMATOUS REPLACEMENT

This refers to the 'compensatory venous plexus in the hilum of the liver, recalling an angioma by its appearance' (Langdon Brown 1901), which develops to replace the lost portal blood flow in both types of obstruction and indeed, even where compression of the portal vein is entirely extrinsic. It is a misnomer to say that the portal vein is 'transformed' (Parker and Seal 1955) into a cavernoma. All the small veins of the gastrohepatic omentum



FIG 20

Case No 129 Double portal venogram in a case of appendix abscess with portal pylephlebitis. Remnants of the portal vein can be seen within the developing cavernoma.

gallbladder, and biliary passages take part in this replacement as pointed out by Sappey in 1883. Those on the surface of the common bile duct appear to be the most constant. The process can occasionally be seen in its very beginning in cases of cirrhosis with portal stasis even when there is no thrombosis (Fig 19). A later stage is shown in Fig 13b and a still later in Fig 20. Recognition of the part played by the biliary veins in the replacement is of vital importance surgically. The channels may become so large and turgid with hypertensive portal blood that the common bile duct itself becomes displaced from its expected position and enveloped in the vascular mass (vide Fig 18). The surgeon may be tempted to dissect into the cavernoma in the hope of procuring a vein suitable for porta-caval anastomosis. Haemorrhage

is likely to be profuse and in such circumstances the common bile duct can only too easily be caught up in a ligature

COMPRESSION, INVASION, ETC., OF THE PORTAL VEIN

The list of causes as set out on the table could be multiplied considerably. Indeed a recent case No 251 showed obliteration of portal and splenic veins by a carcinoma of the pancreas (Fig 11). Child (1952 & 1954) discusses at length the implications of such an obstruction and has shown that involvement of the portal vein does not necessarily imply that the tumour is inoperable in that the human subject can sometimes survive excision of a segment of his portal vein. Another relatively common condition not yet encountered is a cyst of the pancreas (Thompson, Hurwitz and Yesner, Das and Basu).

The diagnosis of these cases often remains speculative until demonstrated at operation because of their astonishing complexity and because of the intermingling of cause and effect. Case No 74 is an example which is in my experience unique. She had primary splenic anaemia. The spleen was very large and was situated behind the left kidney which had in consequence been displaced forwards and to the right until it finally lay to the right of the midline behind the foramen of Winslow. Here it compressed the portal vein. The portal pressure was 405 mm water before and 110 mm after relief of the compression by splenectomy. The spleen had in effect contributed to its own congestion.

Case No 47 had arterial hypertension and a water hammer pulse with a loud machine murmur over and below the liver. Gastro-intestinal haemorrhage occurred from time to time and came from oesophageal varices. Pycnography showed a distorted and displaced right kidney. This and the portal vein were therefore explored through a horizontal incision. The kidney contained an enormous cirroid aneurysm and a hypernephroma which compressed the portal vein. Nephrectomy relieved the portal hypertension and the hypernephroma has not recurred in nearly six years even though it protruded into the renal vein.

Splenic arteriovenous aneurysm has also been reported by Welch (1950) as a cause of portal hypertension. It certainly elevates the pressure but the effects it produces are not comparable with those found when the portal vein is obstructed and the flow reduced in speed. It probably should be regarded as an example of forward pressure rather than back pressure. The treatment as with other cases of compression and invasion of the portal vein is the removal of the cause.

CHAPTER V

INTRAHEPATIC PORTAL OBSTRUCTION OR CIRRHOSIS HEPATIS

OF the 250 patients under review 218 suffered from cirrhosis hepatis (see table, Fig 21). An attempt has been made to discover the cause of the cirrhosis in each case, but the limitations and difficulties of such an investigation are fully recognised. The deductions are presented in the knowledge that many will have to be reviewed in the light of new discoveries and as the ultimate fate of the patients is known. For example, one patient has died of haemochromatosis with diabetes since the analysis was completed.

The largest group undoubtedly is that of the so-called *idiopathic* or *cryptogenic* (Dible, 1951) cirrhosis in which there is apparently no antecedent disease or cause to account for the hepatic fibrosis. In this group there were ninety six patients (44 per cent of the cirrhotics). Most of them were typical examples of Laennec's cirrhosis but some were included because they defied classification.

One such, a woman aged 60 (Case No 110, Fig 22), presented with myelosclerotic anaemia and intrahepatic portal obstruction with calcification and myeloid metaplasia within the spleen as described by Turnbull in 1936. Following splenectomy and porta-caval anastomosis she became plethoric and two years later developed myeloid leukaemia. The liver showed some increase in the periportal connective tissue and extramedullary myelopoiesis.

Two children have been encountered (Figs 23, 24 and 25) who have exactly answered the description given by Hilton Fagge and Hale White in Osler's 'Medicine' that 'the liver could be grasped in the hand and formed a rounded small organ'. Both have been classified as idiopathic (congenital). It is possible however that they inherited the virus of infectious hepatitis from their mothers during pregnancy and they may be examples of foetal hepatitis (Dible *et al.* 1954, Moncrieff 1953).

In forty five or 20.6 per cent there had been a history of infective hepatitis in the past and in the majority of these microscopical examination of the liver appeared to confirm the clinical diagnosis. However, in some patients the attack of pyrexia associated with jaundice could have been an exacerbation of hepatitis in an already damaged liver. In others attacks of apparent hepatitis had occurred in the presence of other conditions leading to cirrhosis and it was difficult to know which cause was the more important. Two cases of syphilis for example had had serum hepatitis. In others an attack of typical infective hepatitis was later followed by cirrhosis hepatis which developed into the so-called primary biliary cirrhosis as defined by Sherlock

INTRAHEPATIC PORTAL OBSTRUCTION

CIRRHOSIS HEPATITIS

INTRA-HEPATIC OBSTRUCTION

Total 218 Patients 81.2% of all.

	BIOPATHIC	POST INFECTIONE	ALCOHOLIC	P.O.W.	BILIARY	ARSENIC	COLD	CCl ₄	CHOLANGIO- HEPATITIS	TOXAEMIA OF PREGNANCY	SYPHILIS	CARDIAC
MILD Total 88 cases	20	8	1	2	2		1	1	1			
Females 11												
Average age 47 years												
Males 22	60.8%	18.0%		6.0%						1		
Average age 32.4 years												
MODERATE Total 75 cases	36	12	9	6	3	3	1		1	4		
Females 33												
Average age 44.6 years												
Males 42	48.0%	18.0%	12.0%	8.0%	4.0%							
Average age 42.7 years												
SEVERE Total 110 cases	40	31	18	8	11	1			1	1	3	
Females 46												
Average age 50.1 years												
Males 64	30.3%	24.5%	18.4%	7.5%	10.0%							
Average age 47 years												
TOTALS	96	45	27	15	16	6	1	1	1	2	5	3
218 cases	44.0%	20.6%	12.4%	6.9%	7.3%	2.7%	0.5%	0.5%	0.5%	0.9%	2.3%	1.4%

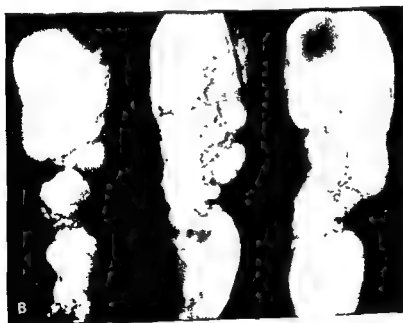
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PORTAL HYPERTENSION

(1955) and by MacPhee (1956). The situation is therefore confused, but the general conclusion to be drawn is that in this country less than one-quarter of the cases of cirrhosis hepatis are post-infective in origin.



FIG 22
Case No 110 A The
spleen and gastric
varices on abdominal
X ray (Barium meal)
B The spleen itself
Calcification in con-
gestive splenomegaly
(myelosclerosis and
cirrhosis)



Twenty-seven (12.4 per cent) gave a clear and indubitable history of *chronic alcoholism* without any other obvious cause. The proportion is small compared with most series that are published because medical treatment is

INTRAHEPATIC PORTAL OBSTRUCTION



FIG. 23

CASE N. 149. Venogram and photograph of the cholecyst of a boy aged 5

PORTAL HYPERTENSION

(1955) and by MacPhee (1956) The situation is therefore confused but the general conclusion to be drawn is that in this country less than one quarter of the cases of cirrhosis hepatis are post infective in origin



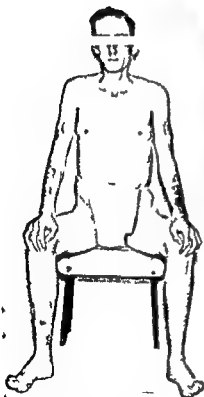
FIG. 22
Case No 110 A The
spleen and gastric
varices on abdominal
X ray (Barium meal)
B The spleen itself
Calcification in con-
gestive splenomegaly
(myelosclerosis and
cirrhosis)



Twenty seven (12.4 per cent) gave a clear and indubitable history of *chronic alcoholism* without any other obvious cause. The proportion is small compared with most series that are published because medical treatment is

INTRAHEPATIC PORTAL OBSTRUCTION

often of great benefit and it is this group that the surgeon usually does not see. Many other patients consumed alcohol from time to time but not in quantities which were excessive or which could have been considered likely to cause liver damage in the absence of other predisposing conditions.



B



A

Fig. 6

Prisoner-of-war Far East

Case No. 245. On release and ten years later following fifteen months of intensive medical therapy for ascites.

Fifteen patients (6.9 per cent) had been prisoners-of-war: fourteen in the Far East, one in East Prussia. All had suffered from severe protein deprivation (Himsworth, 1950) and most had had many other associated diseases: infective hepatitis, duodenal ulcer, multiple attacks of malaria, beri-beri.

PORTAL HYPERTENSION

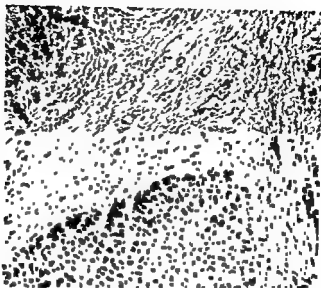


FIG 24

Case No 149 Photomicrograph of the same liver to show the extreme fibrosis in the portal tracts and healthy liver cells in the proliferating lobules



FIG 25

Case No 81 The cirrhotic liver of a girl aged 5 which was so mobile that it could be delivered on the abdominal wall

INTRAHEPATIC PORTAL OBSTRUCTION

calculus can usually be removed without difficulty and this should be done before deciding whether or not the portal hypertension needs operative treatment (Cases Nos 53 and 277). Removal of biliary congestion usually allows vascular congestion to improve. With a common duct stricture on the other hand the situation is different. Periductal adhesions are probably massive and very vascular. Cole, Ireneus and Reynolds (1955) found portal hypertension present in twenty nine out of 122 cases of common duct stricture. It led to much haemorrhage when attempts were made to reconstruct the duct. Some operations even had to be abandoned. They came to the conclusion that the portal hypertension should be relieved by lienorenal anastomosis as a first stage the duct being repaired later. In these cases porta-caval anastomosis is not likely to be possible because of insuperable difficulties of access and associated thrombosis (Fig. 17).

Congenital atresia of the biliary passages was present in two boys of 6 and 3 years of age (Fig. 27). The child illustrated died of haemorrhage from a gastric ulcer but his condition was hopeless before this terminal catastrophe. Postmortem a hepatoma was discovered. During the whole of this investigation only one other hepatoma has been encountered.

Primary biliary cirrhosis was also encountered eight times all in women the youngest 38 and the eldest 67 with an average age of 54 (Fig. 28). The disease though severe is surprisingly prolonged. MacPhee (1956) gives a good description.

There were eight cases of *toxicopathic cirrhosis* apart from alcohol.

Six patients or 2.7 per cent had taken *inorganic arsenic* therapeutically for many years usually in the form of liquor arsenicalis added to some mixture. Two patients had taken it as a tonic one to give relief from the irritation caused by dermatitis herpetiformis and three for the mitigation of epilepsy. It was often difficult to discover exactly how conscientiously these medicines had been taken and for how long. The period had never been for less than four years and in only one case had it been for more than fifteen. In all these patients there was unequivocal evidence of chronic arsenic poisoning either in the form of raindrop pigmentation (Fig. 117) or keratosis punctata (Fig. 29) or both. Two have recently died at the ages of 57 and 40 of carcinoma of the bronchus and two others are developing multiple epitheliomatous areas on their skin (Figs. 29 and 117c). The average age of this small group of patients is 43 and it seems remarkable that four out of six should develop cancer of skin or lung unless the arsenic is to blame.

Himsworth, Wade and Frazer and Aukland all emphasise the danger of chronic arsenicalism and a recent *Lancet* editorial on Arsenic in the House demonstrates how serious a threat even the unconscious ingestion or inhalation of arsenic dust from house paints can be. Inorganic arsenic should not

PORTAL HYPERTENSION

tropical ulcers, starvation oedema, etc (Fig 26) They constitute a particularly distressing group

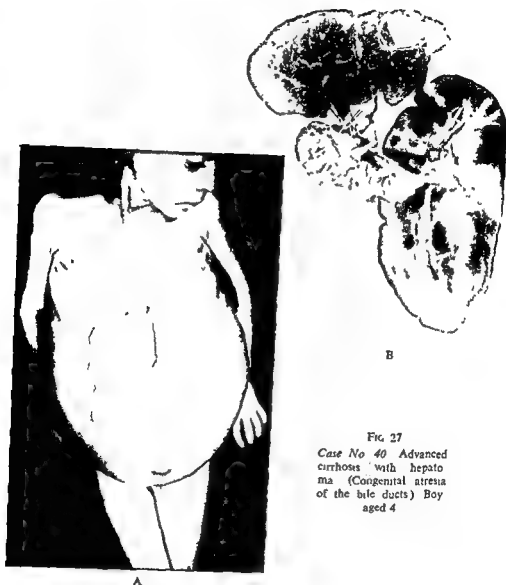


FIG. 27
Case No 40 Advanced
cirrhosis with hepato
ma (Congenital atresia
of the bile ducts) Boy
aged 4

Biliary cirrhosis was present in sixteen patients, though others may have to be added to this list

In eight the cirrhosis had developed following long standing biliary obstruction and was of the *secondary type* (Boyd, 1954, Ellis and Holman, 1955) Biliary stricture or calculus or both were present in six (Fig 120) A

INTRAHEPATIC PORTAL OBSTRUCTION

In addition to these six patients there were three others who had taken arsenic. One gave a clear-cut history of infective hepatitis which had been inadequately treated and which had been followed by recurrent attacks of icterus and general ill health. This patient also suffered from dermatitis herpetiformis, for which he took inorganic arsenic from time to time, but not

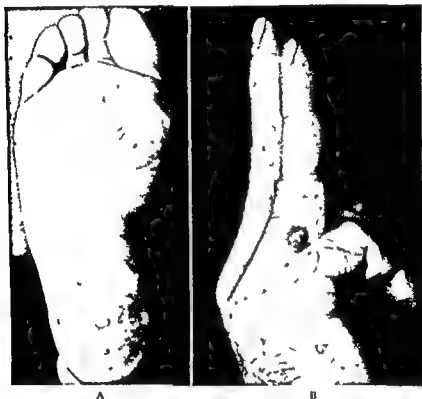


FIG. 29

Case No. 104. Keratosis punctata of inorganic arsenic poisoning with the development of epithelioma *in situ*.

in doses which could be considered of aetiological significance. Another (Case No. 119) had suffered from splenic anaemia from childhood. This had been treated with a 'blood tonic' containing inorganic arsenic. The spleen was then removed. Finally it was discovered that the cause of her Banti's syndrome was a congenital obliteration of the portal vein (Fig. 8) and superimposed upon this she has a fine granular cirrhosis of the liver which was presumed to be the result of the arsenic. In the present analysis she is considered primarily as a case of extrahepatic portal obstruction. The third patient

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be given even in its normal dose for more than short periods at a time, though its therapeutic value cannot be denied. It is a strong poison and toxic in many



FIG 28

Case No. 246 A B Portal venogram and cholangiogram in a patient with advanced primary biliary cirrhosis. To show lobulated distortion of liver pattern and diversion of blood up oesophagus and an unobstructed biliary tree in spite of the jaundice. C D The same patient before and two and a half months after porta caval anastomosis to show elimination of ascites with reduction of girth by 9 inches.

different ways. The dangers of its administration should be more widely recognised particularly its effect on the liver and its strange action as a carcinogenic agent.

INTRAHEPATIC PORTAL OBSTRUCTION



FIG. 30

porta-caval
The cavity
invaded and
cured

referred to in this respect is described under the heading of carbon tetrachloride

A solitary case of cirrhosis hepatis following three courses of sanocrysin treatment for ankylosing spondylitis deformans was encountered (Case No 159). The total amount of *metallic gold* that had been administered was 1.75 gm (in 3.88 gm of gold salts) over a period of two years. Each of the three courses had been carried to the limit of toxicity, with the production of icterus. No other cause for his cirrhosis could be discovered though he had also been given the 'Russian treatment'—the dropping of infected material from a case of infective hepatitis into the nostrils—but it had not taken. His spondylitis was not affected by all this. Porta-caval anastomosis was done for Banti's syndrome, with temporary improvement in the patient's condition. He then began to deteriorate and died of a primary carcinoma of the liver eighteen months after operation (Fig 30). The amount of gold present in his liver, as found in the fragment removed at operation for microscopical examination and postmortem, was considerable, 1.8 micrograms in 1 gm of liver, whereas the amount in the carcinoma itself was negligible. These estimations were done using the method of radioactive spectrometry by Dr R. A. Allen of Harwell.

Attempts to trace other cases of gold cirrhosis and to discover whether patients with toxipathic hepatitis due to heavy metals are more likely to develop carcinoma than other cirrhotic patients have been without any result. Gold is not usually considered as a cause of cirrhosis, though it is mentioned as a possible aetiological factor from time to time, e.g., MacPhee.

Carbon tetrachloride was considered to be the cause of cirrhosis in one patient, a woman who had worked for fifteen years in the chemical solvents department of a manufactory (Case No 100). Towards the end of her time in this employment she had become anaemic and for this had taken a blood tonic containing inorganic arsenic, but the quantities of arsenic had been small and the duration of its administration short. She, too, has developed an epidermal neoplasm of multicentric origin.

Cholangio-hepatitis—The solitary case of cholangio hepatitis is that of a young man who has had recurrent attacks of jaundice with pyrexia since boyhood. Operative cholangiography has demonstrated a normal biliary tree and microscopy of the liver a very mild periportal fibrosis. His condition now is so good that he requires no treatment.

Toxaemia of pregnancy—Two patients were perfectly fit until they developed toxaemia of pregnancy. In one (Case No 24) the liver improved with the delivery of the baby, but deteriorated again during a subsequent pregnancy, which was therefore terminated. In another the cirrhosis appeared

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to start with toxæmia of pregnancy, yet the woman now suffers from a condition which resembles primary biliary cirrhosis

Syphilis (McCrae and Caven, 1926, Osler, 1944, Lazzari and Rack, 1951, and Alergant, 1956) was certainly the aetiological factor in two patients. One responded to medical treatment (reported by Nicol and Terry, 1951); but the other (Case No. 205, Figs. 31, 32, and 80) required a spleno-renal anasto-

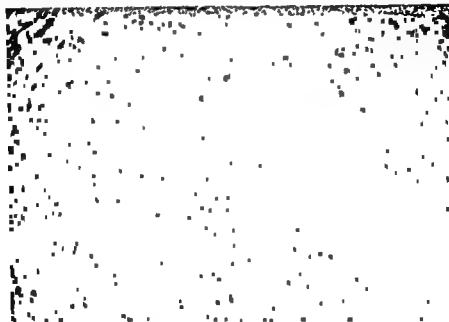


Fig. 32

Gummatous Hepatitis

Case No. 205. The microscopic appearance of the liver shown in Fig. 31a

miosis for intractable ascites. It has been possible in this second case to carry out a complete investigation including microscopical examination of the liver. The liver is lobulated, cirrhotic, scarred, and contains gummata. In addition to these two incontrovertible cases, there were three others in which there had been no aetiological factor prior to the syphilis. They had, however, received treatment for syphilis and the possibility of serum hepatitis acquired during the course of therapy cannot be excluded (Dible *et al.* 1943).

Chronic cardiac disease was present as the sole aetiological factor in three patients and they have been considered as cases of cardiac cirrhosis.



FIG 11

Case No 205 Venogram (A) and photograph (B) of syphilitic liver, 'hepar lobatum'. The portal vein is partially thrombosed, collateral biliary vessels are distended

INTRAHEPATIC PORTAL OBSTRUCTION

therefore, the age increased with the severity of the condition. The preponderance of males in the moderate and severe groups is due to the greater number of male alcoholics and the prisoners-of-war.

ASSOCIATED CONDITIONS

Peptic Ulcer.

Peptic ulcer was present in eighteen of the 218 cases of cirrhosis hepatis, an incidence of 8.25 per cent. This is considerably in excess of the incidence in the population generally but agrees with the 10 per cent noted by Welbourn (1952), and Fainer and Halsted (1955) observe the same tendency, which is probably due to congestion of the mucous membrane and a reduction in its powers of resistance (Baronofsky). It is easy to appreciate the difficulties to which an associated ulcer may give rise (see the 'Treatment of Acute Haemorrhage').

Diabetes.

Diabetes has been encountered in six patients. In four it was known to be present before operation was undertaken but in two it developed as a complication following operation (Cases Nos. 223 and 187), and one of these patients has since died in diabetic coma. In a condition such as cirrhosis hepatis, where circumstances may arise in which the treatment is by the administration of relatively large quantities of glucose the possibility of an associated diabetes must always be borne in mind. (Subsequent developments in Case No. 223 and the study of Case No. 292 have shown that haemochromatosis was present and probably accounted for the cirrhosis and the diabetes in both patients. In Case No. 223 it was not possible to confirm its presence by liver or skin biopsy nor was the serum iron elevated.)

Nephritis.

The association between hepatic and renal disease has long been recognised though probably on faulty evidence. In the 218 cases of cirrhosis definite nephritis was present in only three patients. In two it has led to the patient's death and in the third it is of such severity as to preclude any operation for the relief of portal hypertension.

Kinnear Wilson's Disease.

Hepatico-lenticular degeneration was encountered in one patient, a girl of 13, and she presented with haematemesis due to portal hypertension resulting from cirrhosis hepatis and without neurological signs. Liver function tests indicated considerable derangement. After a lienorenal anastomosis the liver improved up to the time that the cerebral deterioration became serious and led to death (Case No. 65).

CLASSIFICATION OF CASES OF CIRRHOSIS HEPATIS ACCORDING TO SEVERITY OF THE DISEASE

(see Fig 21 p 27)

The 218 patients with cirrhosis have been classified into three categories of severity mild numbering 33 moderate 75 and severe 110 The classification was adopted to simplify decisions concerning treatment and assessment of prognosis

Definitions

1 *The mild cirrhotic* is a patient in whom there are virtually no symptoms or signs of a diseased liver and in whom the liver function tests are not clearly distinguishable from normal The fact that the patient has cirrhosis only becomes evident when he is operated on for portal hypertension or for some other condition which may not be associated with the liver at all The cirrhosis in every case has been of a finely granular type Twenty of the thirty three patients (60.6 per cent) suffered from the so-called idiopathic type

2 *The moderate cirrhotic* is a patient who has definite clinical evidence of liver disease and in whom the liver function tests are undoubtedly deranged though not to any extreme extent The liver is not in a state of failure Half these patients were of the idiopathic type

3 *The severe cirrhotic* is the patient whose liver is failing 110 or more than half were of this type They are very ill usually with gross ascites and in a state where operative intervention without careful pre operative preparation would be fatal Sometimes associated diseases such as diabetes or nephritis compelled a patient to be down graded into this category of poor prognosis though the liver was not very badly diseased Any patient with grossly deranged liver function tests as judged by a serum albumin of less than 3.2 gm / 100 ml was placed in this group Only forty (or 36 per cent) were of the idiopathic variety twenty seven (or 24.5 per cent) had had infective hepatitis in the past eighteen (or 16.4 per cent) were alcoholics eight had been prisoners-of-war and eleven were biliary either primary or secondary

This analysis indicates that a cirrhosis which develops without known predisposing cause is likely to be of a milder type or conversely that a cirrhosis with definite origins in illness is more likely to be severe or progressive Prognosis depends on severity rather than type

The age and sex incidence in these different groups is as follows of the thirty three mild cirrhotics eleven were female with an average age of 47 and twenty two male with an average of 32.4 years of the moderate cirrhotics there were thirty three females average 44.6 years and forty two males average 42.7 years in the severe cirrhotics there were forty six females average 50.1 years and sixty four males average 47 years On the whole

CHAPTER VI

THE EFFECTS OF PORTAL OBSTRUCTION

DIVERSION OF PORTAL BLOOD INTO THE SYSTEMIC CIRCULATION

OBSTRUCTION to the flow of portal blood leads to its diversion along the normal and abnormal communications that exist between portal and systemic venous circulations. In 1835 Professor Retzius of Stockholm demon-



FIG. 33

Veins of Retzius

Case No. 76. A. In paraduodenal fossa resembling a cirroid aneurysm. B. In ligament of renal vein a very capillary mesh.

strated numerous small vessels linking the veins of the duodenum and colon with the inferior vena cava and joining up on the left with the renal vein. He also showed vessels passing from the rectum to the spermatic plexus. His discovery was accidental, while preparing specimens by injection of the portal vein, and he refers to 'les injections heureuses'. The retroperitoneal veins described by Retzius are well demonstrated in Figs. 33 to 37.

The two latter demonstrate graphically that blood from the spleen can be diverted in great quantities into the systemic circulation. If the spleen can

Thrombosis (Figs 14, 17, 20, and 48)

Extensive portal and splenic vein thrombosis has been found in twenty four out of the 218 cases (11 per cent). It does not include cases of 'chronic sclerotic endophlebitis' (Banti) or phleboscclerosis, in which the wall of the vein is thickened and occasionally very hard, and which may be regarded as a condition comparable with arteriosclerosis (Whiteley). Dock and Warthin (1904) described a case where the portal vein had even become calcified.

Thrombosis may be regarded as the addition of an extrahepatic obstruction to an already congested, turgid, and static portal tree. Stasis, splenectomy, infection, and polycythaemia predispose to the condition (Graham Bryce 1932). It adds materially to the difficulties of diagnosis and treatment (Hunt and Whittard, 1954, Hunt, 1956).

Ulcerative Colitis.

Ulcerative colitis was associated with cirrhosis in two cases, No 178 a man of 48 with advanced idiopathic cirrhosis, and No 246, a woman of 46 with advanced primary biliary cirrhosis. Such cases must be judged individually (see Welbourn), and, when circumstances demand, operation for ulcerative colitis may precede or follow porta caval anastomosis. The association of the two diseases may be more than a coincidence.

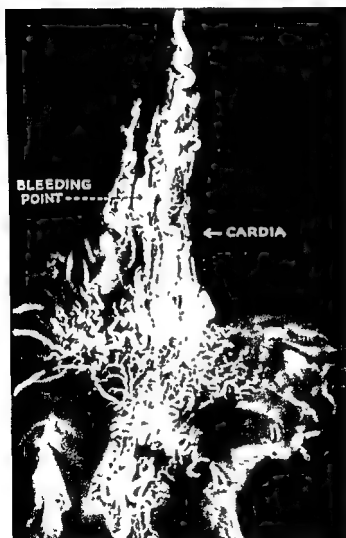


FIG. 40

Case No. 89. Photograph—X ray of stomach and oesophagus in which the veins had been injected with bismuth suspension postmortem to show continuous nature of gastro-oesophageal varices (Smith Rodney (1954). *Progress in Clinical Surgery*. London J & A Churchill Ltd.) (Royal College of Surgeons Museum)



FIG 38

Case No 164 Portal venograms taken on different occasions and at different angles on the same patient. A shows diversion of blood along a large left gastric vein to oesophageal varices and to a large diaphragmatic vein which drains (B) into the phrenic vein (arrowed) and so into the superior vena cava. (The portal vein and its left branch are long and thin (A))



FIG 39

Case No 165 Portal to pulmonary venous anastomoses. Vessels can be seen passing from turgid diaphragmatic veins into adherent lung

EFFECTS OF PORTAL OBSTRUCTION



FIG. 42

Case No. 240 Oesophageal and peri-oesophageal varices.

systems. They also communicate with the *venae comites* of the vagus nerve (Fig. 44) and, as mentioned above, with the diaphragmatic plexus.

This massive and dangerously exposed plexus is both gastric and oesophageal, as can be seen in Figs. 40 and 45. Doubt as to the nature of the gastric filling defects on barium swallow can be dispelled by comparison with the venogram, which sometimes appears like the negative of the barium swallow (Figs. 46 and 47). Occasionally gastric varices may be present alone. Lastly, oesophageal varices are said to exist as a congenital anomaly (Taylor and Egbert).

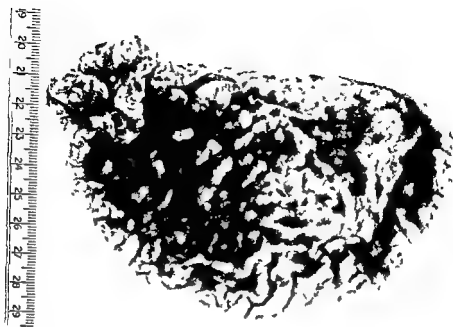


FIG 41

Case No 6 Proximal gastric resection. The resected specimen showing pericardiac varices and multiple deep mucosal erosions (St Bartholomew's Hospital Museum)

The most important of all points of contact between portal and systemic circulations is at the lower end of the oesophagus and cardiac end of the stomach where there is a plexus of veins within the submucous layer with free communication past the cardiac sphincter (Fig 40). Some of the veins are small and lie immediately deep to the lamina propria (Butler, 1951, Kegaries 1933). They can be laid open to the lumen by shallow erosions. Larger veins usually lie deeper in the submucosa but they too are liable to burst into the lumen, sometimes with the loss of enormous quantities of blood (Fig 41, Case No 6). The submucous veins communicate with veins on the surface of the oesophagus (Figs 42 and 43) and drain into the azygos and hemiazygos

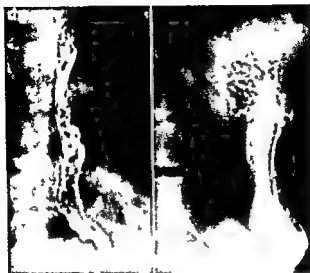


FIG. 43

Case No. 193. Oesophageal and gastric varices in a case of extrahepatic portal obstruction.

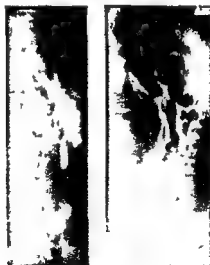


FIG. 44

Case No. 102. Barium meal and venogram to show gastric varices.

PORTAL HYPERTENSION



FIG 43

Case No 247 Peri oesophageal varicosities seen through a thoracotomy incision



FIG 44

Case No 278 Portal venogram showing diversion of portal blood up the vasa nervorum of the left vagus just visible alongside the vertebral column

EFFECTS OF PORTAL OBSTRUCTION

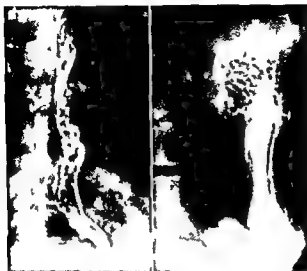


FIG 45

Case No 195 Oesophageal and gastric varices in a case of extrahepatic portal obstruction



FIG 46

Case No 102 Barium meal and venogram
show gastric varices



FIG 43

Case No 247 Peri oesophageal varicosities seen through a thoracotomy incision



FIG 44

Case No 278 Portal venogram showing diversion of portal blood up the vasa nervorum of the left vagus just visible alongside the vertebral column

EFFECTS OF PORTAL OBSTRUCTION

Striking communications are found along the falciform ligament to the anterior abdominal wall, usually by way of an umbilical vein which has failed to be obliterated (Figs 48, 49 & 50) Butler (1954) suggests that obliteration is by contraction and fibrosis, which may not occur if the normal passage of portal blood is blocked. The blood flows from a dilated portal space at the



FIG 48

Case No 187. Portal venogram in a case of moderate cirrhosis to show large patent tortuous umbilical vein (Cruveilhier Baumgarten syndrome) partial thrombosis of portal vein and portal stasis. Speed of flow 2 cm per second

origin of the left branch of the portal vein within the liver back to the anterior abdominal wall. Pegot observed the condition in 1832, Cruveilhier published further details of Pegot's case in 1835 and Baumgarten gave a full description in 1891. The condition is now known as the Cruveilhier Baumgarten syndrome and has been encountered in five cases of cirrhosis and in the two with normal livers described on p. 25. An associated thrombosis is common (Fig 48). It must be that a failure of the umbilical vein to obliterate at birth is a relatively frequent occurrence, which is often missed because the vein is unobtrusively collapsed in the postmortem and dissecting rooms.



A



FIG 47
Case No 239 Gas
tric varices shown
by venography (A)
and by barium
meal (B)

EFFECTS OF PORTAL OBSTRUCTION



FIG 50

Case No 229 Portal venogram to show Cruveilhier Baumgarten disease. Sluggish tortuous umbilical vein emptying into a dilated superior ep gastric vein (lower picture). There was no caput medusae. Portal venous pressure only 140 mm water. (See Fig 56)



FIG 49

Case No 141 Cruveilhier
Baumgarten syndrome Cir
rhotic liver large tortuous
umbilical vein anastomos
ing with inferior epigastric
vein (See Fig 52) (IV^e
Congres de Gastro Entero
logie (1954) L'Hyperten
sion portale Le Duple
Syndrome Paris, Masson
III Cie) (Fig 49a)

JULY 1968

Photostat reproduction of one of Sappey's drawings illustrating the anastomosis between left branch of portal vein and left inferior epigastric vein (Sappey (1883) *J Anat Physiol*)

PORTAL HYPERTENSION

The peripheral connection of this umbilical hepatofugal flow may be to superior (Fig 50) or inferior epigastric veins (Figs 48 and 49) or to the subcutaneous plexus where it gives rise to the famed caput medusae of cirrhosis hepatis so named by Marcus Aurelius (Fig 51) Budd (1845) considered



FIG 51

Case No 125 Caput medusae: Primary carcinoma of liver with portal vein compression

that these veins answer a good purpose and it is worth comparing one of Sappey's drawings (Fig 52) with Figs 48 and 53 Sappey (1883) appreciated the value of this collateral flow in diverting blood when it could not traverse the liver He showed that the flow was *du haut en bas et non de bas en haut* and regarded it as *un signe favorable* in considering *cirrhose ancienne et incurable*

EFFECTS OF PORTAL OBSTRUCTION

inefficiency in relieving a clinical state it must be realised that each channel serves some function and that none should ever needlessly be destroyed

The most exposed and at the same time the most protected of all portal to systemic venous communications connect the rectum with the other pelvic plexuses—one of the groups demonstrated by Retzius. The rectal end of these communications is in the submucosa yet haemorrhoids are probably no more common in cirrhosis than in the population at large. In the present series only one haemorrhoidectomy has been necessary. The patient was a man who had had piles for many years before starting to take an arsenical tonic which was the cause of his cirrhosis. On one occasion a patient attending the general surgical clinic for piles was found to have Banti's syndrome. His piles responded well to injection treatment.

For two years all patients with portal hypertension were sigmoidoscoped without the discovery of anything of significance. It seemed reasonable to conclude that the muscular power of the sphincter ani and the pressure within the rectum was such that even the most hypertensive of superior haemorrhoidal veins would be compressed and that piles could probably not be caused by portal hypertension though pre-existing piles might be aggravated. Occasionally dilated mucosal channels could be seen extending up the sigmoid colon but the findings were never conclusive or of any value.

The attachment of organs draining into portal and systemic circulations has long been known to cause communicating channels to grow across between the two particularly when omentum is stitched to the abdominal parietes—the Talma Morison operation. It can be seen to a greater or lesser extent on every occasion that a second abdominal operation is done in the presence of portal hypertension but much more so when the liver is cirrhotic than when the obstruction is purely extrahepatic. The channels are even less efficient than those present or developing in the normal peritoneal folds of contact but they also should be preserved in the belief that they contribute a little to portal decompression. The difference in the extent of these communications between patients with cirrhotic and normal livers is sometimes so noticeable that it suggests the possibility that abnormal biochemical substances play a part in the stimulus to vessel formation. Enlargement of existing channels seems to be a simple response to increased pressure and stasis whereas the exuberance of capillary proliferation with subsequent enlargement into appreciable veins occurs to a much greater extent in the presence of a deranged liver.

The Budd Chiari syndrome has not been encountered. In this condition the hepatic veins are obliterated, with or without involvement of the inferior vena cava. Excellent illustrations have been published by Jonas and Lawrence. Communications develop along the ligaments of the liver carrying blood in a centrifugal direction, the 'hepatofugaler Pfortaderkollateralen' of Chiari.

It has been proved above, *a propos* Case No. 249 Fig. 36, that natural portal to systemic venous communications may at times be effective in decompressing the portal tree, but far more often they are worthless. The flow



FIG 53

Case No. 151. Cruveilhier Baumgarten syndrome to show sluggish flow in umbilical vein. Speed of flow measured by the radioactive sodium method in portal vein 4.5 cm per second in umbilical vein 1.5 cm per second.

along the umbilical vein of Case No. 151 (Fig. 53), for example, was found to be 1.5 cm per second, while the flow in the portal vein was 4.5 cm per second before and 8 cm after porta-caval anastomosis. Bergstrand and Ekman (1955) have also demonstrated visually the slow flow in collateral channels.

It is not necessarily the size of the vessel that is important. It may be very large, but what makes it inefficient is that it is often tortuous or kinked, its lumen varies in size, it passes through the muscular wall of the abdomen, and it is liable to be compressed and obliterated completely by muscular contraction or increased intra-abdominal pressure. Jean Auvert (1955) supports this opinion. Beswick and Butler, from the anatomists' point of view, question the value of porta-caval anastomosis when huge collaterals are already present but the inefficiency of these channels is proved by the event of haemorrhage.

The difference between the artificial and the natural channels is that the man-made stoma is a direct, unkinked, protected sluice from the bottom of the pool of portal blood. The spontaneous channel is no more than an overflow which only exists because of the portal stasis. Yet, while condemning their

EFFECTS OF PORTAL OBSTRUCTION

sequence A graph (Fig 54) has been drawn up to form the basis of the subsequent discussion

THE NORMAL PRESSURE

The left hand column of Fig 54 shows the pressures found in twenty six patients with normal portal circulations The highest was 210 mm water, the average exactly 100, and the lowest 40 (in the absence of arterial hypotension) This agrees with the findings of others (Whipple, 1945, Welch,

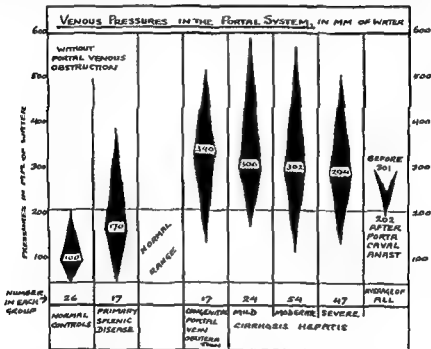


Fig 54

1947, Milnes Walker, 1952, Gray, 1951, Taylor and Egbert, 1951, Santy and Marion, 1951) The average age of the control patients was slightly higher than the average of those with portal obstruction

FACTORS INFLUENCING THE PORTAL VENOUS PRESSURE

The portal venous pressure can be affected in many ways and much must be taken into consideration in assessing each reading First and foremost it depends on the systemic arterial pressures Herrick showed in 1907 that in the normal liver a 40 mm Hg rise of arterial pressure leads to a 1 mm

CHAPTER VII

THE EFFECTS OF PORTAL OBSTRUCTION

HYPERTENSION AND STASIS

THE haemodynamics of the portal circulation have been studied in this investigation by three methods, firstly, by taking venous pressure readings, secondly, by estimating the speed of flow of the blood in the portal and splenic veins, thirdly, by visualising the flow by means of serial radiography (The technical details of the methods employed are fully described in Appendix I)

The portal pressure has been taken by the direct method, using a cerebrospinal fluid manometer, and no correction has been made for the height of the needle puncture above the level of the portal vein. The position of the patient has been constant in every case and the readings form a comparable series. No attempt has been made to correlate the findings with those obtained by splenic or hepatic puncture or hepatic vein catheterisation (Atkinson and Sherlock, 1954, Paraf *et al*, 1955, Paton, Reynolds and Sherlock 1953, Reumann *et al* 1955). My intention from the beginning has been to study values which are comparable, using the simplest possible method. Every attempt has been made to obtain accurate readings and when there has been doubt as to the accuracy of a figure it has been excluded from the analysis.

The estimation of the speed of flow in the portal vein has been done by observing the passage of radioactive sodium along the vein by means of a double probe pointed scintillation detector coupled to a photomultiplier tube and so to a pen recorder, a method originally devised by Professor Mayneord Dr Belcher, and myself (Hunt, 1954b).

The method of serial radiography has been the simplest possible, two pictures being taken within two seconds of each other on each occasion, whether the diiodone was injected into a radicle of the portal vein or into the spleen itself. A double cassette tunnel was used, placed under the patient on the operating table.

The present study was never intended as a specific investigation, but was evolved to provide information which was considered necessary to enable the correct operation to be done.

PORTAL VENOUS PRESSURES

At first it was expected that an absolute level of normality could be found and that hypertension could be assessed in relation to it. However, there is no such level. Many apparent inconsistencies have been encountered in con

EFFECTS OF PORTAL OBSTRUCTION

information. Accurate diagnosis by serial radiography is essential and the measurement of portal speed may be of great additional value

PORTAL VENOUS PRESSURE IN EXTRAHEPATIC (PREHEPATIC) PORTAL OBSTRUCTION

Seventeen cases of congenital portal vein obliteration have been investigated (column 3, Fig. 54). Most of them were young and all were operated on for haemorrhage. The highest pressure was 520, the lowest 140 mm of water, the average 340. Comparing this average with the average found in normal patients—100—there is no doubt about the hypersensitive state of the group as a whole. In an operation on a patient, however, it is not averages that matter but the individual. The venogram of the patient with a pressure of 140 (Case No. 229, Fig. 50) is used to illustrate the condition of Cruveilhier Baumgarten disease. The diagnosis of portal obstruction with stasis and diversion of portal blood leading to massive haemorrhage was convincingly demonstrated. Yet the pressure is well within the normal range and little above the normal average.

The conclusions are self-evident. (1) That the term 'portal hypertension' is incorrect for certain cases in that portal stasis with diversion of blood up oesophageal varices can be present in association with a normal portal pressure, and conversely, the portal pressure can be markedly elevated without portal obstruction or diversion of portal blood. (2) That a careful and thorough investigation of every case is essential to define the type and extent of the obstruction and stasis so that the correct operation may be done.

Nothing is to be gained by analysing the fall in pressure resulting from venous anastomoses when the initial pressure is little above normal. As may be expected, the higher the initial pressure the greater the drop.

In other types of extrahepatic portal obstruction such as thrombosis or compression of the portal vein by tumour or cyst or invasion by carcinoma, the pressures have varied considerably. The average has been in the region of 300 mm of water. It is in this group that the highest pressures of all have been encountered, two being over 600 mm. When compression alone is the cause of the portal hypertension, it has been found that simple removal of the impinging structure restores the pressure to normal, as illustrated in Case No. 74 in which the pressure dropped from 400 to 110.

PORTAL VENOUS PRESSURE IN CASES OF CIRRHOSIS

The pressures in the three groups of cirrhotic patients, the mild, the moderate and the severe, are set out in columns 4, 5 and 6 of the graph (Fig. 54). The averages of each group are 300 ± 6 and a careful review of all the notes in an attempt to relate the pressures to the symptoms of haemorrhage,

PORTAL HYPERTENSION

Hg rise in portal venous pressure, and that in the cirrhotic liver the relation ship is 6 to 1. At a recent operation on a man with extrahepatic obstruction it was possible to demonstrate a rise of 60 mm water in portal venous pressure following a rise of 30 mm Hg in the systemic arterial pressure, this ratio agreeing well with Herrick's. Each portal venous pressure reading must therefore, be assessed in relation to the blood pressure (Boerema 1954). McMichael (1932) demonstrated the elevation induced by vasoconstrictor drugs.

Portal venous pressure is raised by an increase in intrathoracic pressure. A reading will not be accurate when there is any obstruction to respiration (Alexander, 1951, Taylor, 1954).

Two further matters, not so easy to define, have also been considered. Firstly, if the intestinal canal is in a completely quiescent state at the time of operation as a result of starvation, the pressure will be correspondingly lowered. Secondly, spasm of segments of the jejunal vein has been seen from time to time immediately after the construction of portal to systemic shunts. A pressure reading from a radicle peripheral to such a zone may be disappointingly high.

PRESSURES OBTAINED IN CASES OF PRIMARY SPLENIC DISEASE WITHOUT PORTAL OBSTRUCTION

The figures obtained in seventeen cases of primary splenic disease were as follows: highest, 390 mm water, average, 170, and lowest 50, as set out in column 2 of the graph. Most were above the average of normal. The variations were considerable not only between different diseases but between cases of the same disease. In acholuric jaundice it was as high as 345 and as low as 50. In a case of splenomegaly associated with myelosclerotic anaemia the highest pressure of any unobstructed portal tree was encountered 390 mm water. Without radiographic control this could well have been termed 'indubitable portal hypertension' but there was no stasis and the pressure dropped to 200 immediately after splenectomy. When such pressures are high, the drop has always been marked on removal of the spleen. Other pressure readings have been as follows: in primary splenic anaemia 210, 205, 115 and 90, in Felty's syndrome 225, and in sarcoidosis 90.

It seems that the increased surge of blood to the portal tree raises the pressure a condition which may be called active portal hypertension (Paraf *et al* 1955). It is correctable by removing this source of the blood, which is the spleen. The condition is comparable with splenic arteriovenous aneurysm. Attention must be drawn to this elevation of pressure in the absence of obstruction so that unnecessary porta-caval anastomoses are not done. Perhaps those reported by Dunlap *et al* (1952), and Mino, Murphy, and Livingstone (1949) for sarcoidosis were unnecessary. The reports do not give enough

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drop of 183 mm water (1951) and Jahnke *et al* (1953) a consistent lowering. The average in my series has been 99 mm water from 301 to 202 a figure including many cases in which the pressure was higher after a shunt than before. The inconsistencies of pressure readings and the frequent demonstration of clinical success in the face of apparent hydrostatic failure again prompts the hypothesis that portal hypertension *per se* is not so important as

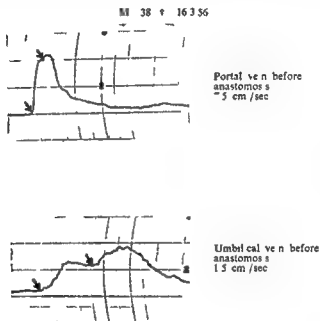


Fig 56

Case No 229 Graphs to show sluggish flow and long persistence of radioactive sodium in the umbilical vein compared with portal (See Fig 50 p 57)

portal stasis It is disappointing when a satisfactory and large stoma has been constructed to find that the pressure within the portal bed has apparently become appreciably raised. With greater experience less reliance has been placed upon the pressure readings than upon the speed of flow through the portal vein in determining whether the shunt was working well or not.

SPEED OF FLOW IN THE PORTAL VEIN

The speed of flow in the portal vein has been estimated side by side with the portal pressure on a number of patients since 1953. The results have been set out on a graph (Fig 55). It will be seen that the average speed in ten control patients who had peptic ulcer or gallstones was 8.7 cm per

ascites, and jaundice did not reveal any significant difference. An earlier analysis (Hunt, 1954b) suggested that the pressure became slightly less in the advanced cases, but this is not noteworthy. It is the variations between similar cases within each group that is astonishing.

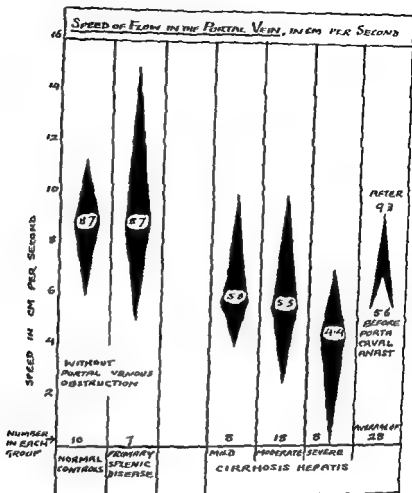


FIG 55

It will be seen that a number of patients have pressures well within the normal range, though never as low as the average of normal. In deciding the correct operative treatment for these patients with the lower pressures individual assessment is again essential.

PRESSURE DROP FOLLOWING PORTA-CAVAL AND LIENO RENAL ANASTOMOSIS

Consistent reduction of pressure has not been obtained as a result of porta-caval or lieno-renal anastomosis although Blakemore records an average

EFFECTS OF PORTAL OBSTRUCTION

indicate the time that it takes for the portal blood to be dispersed. It indicates stagnation within the portal bed as well as a slow flow along the portal vein (Fig 57). After porta caval anastomosis the decompression of the portal bed occurs very much more rapidly than it did beforehand.



FIG 58

Case No 159 Venograms to show portal stasis in a case of moderate cirrhosis. Portal venous pressure 120 mm water. Interval between exposures two seconds in both cases.

The speed of flow has also been measured in the splenic vein. It is on the whole slower than in the portal vein though the variations are considerable. After lieno renal anastomosis the speed of the reversed flow measures about 7.5 cm per second.

second, the highest 11.4, and the lowest 6.0. In seven patients with primary disease of the spleen the average speed was exactly the same, but the spread was greater, the highest being 15.1 the lowest 5.0. In cirrhosis hepatitis the three groups show a decline with advance in the disease. Mild cirrhotics showed a speed of 5.8, moderate 5.5, and severe 4.4. In only two cases was the speed in an individual case of cirrhosis greater than the normal average. The overlap with the normal range is considerable, and the same problems of interpretation apply to an analysis of portal speed as to an analysis of portal pressure. The groups before and after porta-caval anastomosis however

Mc J 10 7 18 5 56

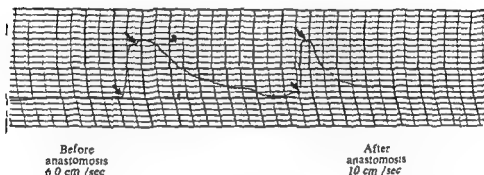


FIG 57

Case No 91 Graph to show slow speed prolonged plateau and lingering disappearance of the radioactive sodium from the portal tree before anastomosis increased speed and rapid disappearance after

showed a constant increase in speed (from 5.6 to 9.3). In six patients in whom the pressure increased materially the speed also increased indicating that the flow through the portal vein was greater after porta-caval anastomosis whatever was happening to the state of venous pressure in the periphery of the portal tree. In only two patients out of twenty-eight has the speed been as low after the shunt as 5.5 and in both these patients the porta-caval anastomosis has thrombosed. The slow speed at the end of operation was therefore an accurate pointer to the outcome.

The graph of portal speeds indicates without a doubt that portal stasis is important as a factor in considering the condition that has come to be referred to as portal hypertension. The portal pressure is usually a reliable guide but there are exceptions which can be very confusing and it is in these that an estimation of the degree of stasis by the use of radioactive sodium or by visualisation of the degree of stagnation by means of serial radiography is of the greatest value.

Radioactive sodium not only gives an estimate of the speed with which the head of the column of blood passes the tips of the probe, but may also

EFFECTS OF PORTAL OBSTRUCTION

kinking or misplacement of the vein or to the position of collateral anastomotic vessels

Thrombosis may confuse the findings, as illustrated by two cases. X rays of the first are shown in Fig. 48, where there is obvious stasis, yet the flow alongside the clot is reasonably fast. The second is a syphilitic patient who has been operated on twice. On the first occasion venography demonstrated the portal vein to be normally patent, the pressure was 290 mm. of water and the speed of flow was 3.5 cm. per second. On the second occasion four months later, the portal vein was shown to have undergone partial thrombosis (Fig. 31A), the pressure was 280 mm. of water and the speed had increased to 9 cm. per second. The channel had become narrowed and collateral vessels had appeared, to give a false impression of speed.

The scintillation detector has also been used to measure accurately the interval between the exposures of the serial radiographs and has thus helped in learning their correct interpretation. It is probable that the measurement of portal speed as a routine investigation can be dispensed with provided that accurate serial X rays can be obtained.

RADIOGRAPHIC DEMONSTRATION OF PORTAL STAGNATION

Radiographic demonstration of portal stasis is illustrated in Fig. 58. The second picture is very similar to the first. The blood extends into the liver and remains almost stagnant in the portal vein. Compare this with Fig. 59, where the blood has emptied itself out of splenic and portal veins within three seconds, the speed of flow being measured at 12 cm. per second.

PORTAL HYPERTENSION

The method needs to be interpreted with care and in relation to the X-ray picture in that it measures the speed of passage of the blood containing

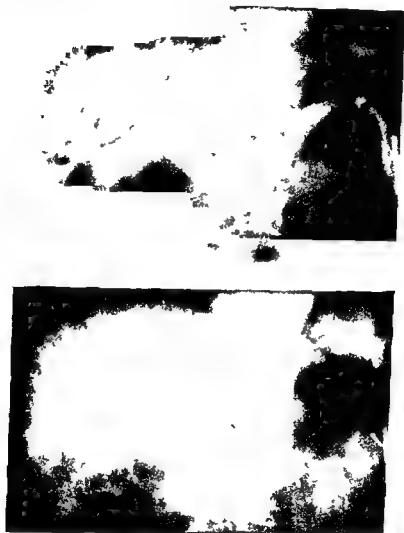


FIG 59

Double splenic venogram in case of splenic anaemia without portal hypertension. The second splenic water

9

radioactive sodium along a certain course and not necessarily through a certain channel. Portal venograms should be available before the instrument is placed in position, otherwise the interpretation may be at fault, due to

CLINICAL MANIFESTATIONS OF PORTAL HYPERTENSION

and obstructed the portal vein an association of symptoms and pathology first pointed out by Langdon Brown in 1901

MODERATE CIRRHOSIS

1 Haemorrhage

In this group the emphasis still remains on the symptom of haemorrhage with its background of hypersplenism but the effects of the haemorrhage become more devastating because of the worsened state of the liver. Whether the haemorrhage is controllable or not the patients suffer far more and take much longer to recover. This additional damage to the liver is due to at least two causes (1) The anoxia of sudden portal blood loss and (2) the absorption of break-down products of blood from the intestine which often leads to the development of a comatose state. In fifty three of the seventy five patients in this group haemorrhage was the initial symptom

2 Ascites

In fifteen patients ascites alone was the initial and major symptom. In addition an appreciable quantity of ascitic fluid was found to be present in nine of the fifty three who complained of haemorrhage

3 Jaundice

Jaundice was a major complaint in four patients

4 Anaemia and general deterioration was the main complaint in three in one it was discovered on investigating his haemorrhoids (This patient died of his first haemorrhage)

ADVANCED OR SEVERE CIRRHOSIS

Ascites and Liver Failure, with or without Haemorrhage

The emphasis in this group is on the deteriorating liver. A hundred out of 110 patients had *ascites* of which forty three had *haemorrhage* in addition. Many had *peripheral oedema*. Of the remaining ten seven presented with haemorrhage as their major complaint which was associated with *jaundice* persistent anaemia or diabetes. Two had severe *anaemia* alone and in one the advanced degree of liver damage was asymptomatic and discovered accidentally at an abdominal operation. This last patient died of his first haemorrhage. Two patients in this group had right hydrothorax complicating and relieving the ascites similar in many ways to the patient described by Emerson and Davies. In both the hydrothorax and ascites improved *pari passu*

Coma

Coma was a common concomitant of haemorrhage in this group. Its initial symptoms are important to recognise—the rather vacant politely unco-operative worried and unsmiling patient who often shows a peculiar flapping

CHAPTER VIII

THE CLINICAL MANIFESTATIONS OF PORTAL HYPERTENSION AND INDICATIONS FOR OPERATIVE TREATMENT

CLINICAL MANIFESTATIONS

THE symptoms and signs of portal hypertension depend upon three things (1) The oesophageal and gastric varices giving rise to haemorrhage, (2) an enlarged and over active spleen causing anaemia and cytopenia, and (3) cirrhosis hepatis. They occur together or separately or in uncertain combinations and it is impossible to foretell exactly what pattern or course will be followed by any one particular patient. The difficulties of diagnosis may be very great, especially when the haemorrhage is severe and the patient desperately ill. Each group will be considered separately, remembering that mild cirrhotics by definition emulate patients with normal livers and are, therefore studied with them.

EXTRAHEPATIC OBSTRUCTION AND MILD CIRRHOSIS

1 Haemorrhage.

Fifty three out of sixty five patients in these two groups presented with haemorrhage as their major complaint. Usually the bleeding could be controlled and the patients recovered normal health within a few weeks. How often a severe haemorrhage causes the death of a patient in these two groups it has been impossible to estimate. One of the earliest patients, while under observation at home, died of his second haemorrhage before medical aid could be summoned. Such a tragic event in an otherwise healthy young man emphasises the dangerous state in which these patients live.

2 Splenomegaly and Splenic Anaemia.

A general state of ill health and anaemia with the discovery of an enlarged spleen on physical examination was the initial clinical picture in five patients including those with myeloid leukaemia and lymphosarcoma.

3 **Accidentally or incidentally** the remaining seven patients were discovered to be suffering from portal hypertension. In two a cirrhotic liver was discovered during the course of operations for appendicitis and duodenal ulcer respectively. In others it was discovered as a concomitant of carcinoma of the stomach biliary obstruction etc. A recent patient, No 251 (Fig 12), was explored on account of persistent anaemia with occult blood in the stools. He was found to be suffering from carcinoma of the pancreas which had invaded

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and obstructed the portal vein an association of symptoms and pathology first pointed out by Langdon Brown in 1901

MODERATE CIRRHOSIS

1 Haemorrhage

In this group the emphasis still remains on the symptom of haemorrhage with its background of hypersplenism but the effects of the haemorrhage become more devastating because of the worsened state of the liver. Whether the haemorrhage is controllable or not the patients suffer far more and take much longer to recover. This additional damage to the liver is due to at least two causes (1) The anoxia of sudden portal blood loss and (2) the absorption of break-down products of blood from the intestine which often leads to the development of a comatose state. In fifty three of the seventy five patients in this group haemorrhage was the initial symptom

2 Ascites

In fifteen patients ascites alone was the initial and major symptom. In addition an appreciable quantity of ascitic fluid was found to be present in nine of the fifty three who complained of haemorrhage

3 Jaundice

Jaundice was a major complaint in four patients

4 Anaemia and general deterioration was the main complaint in three in one it was discovered on investigating his haemorrhoids (This patient died of his first haemorrhage)

ADVANCED OR SEVERE CIRRHOSIS

Ascites and Liver Failure, with or without Haemorrhage

The emphasis in this group is on the deteriorating liver. A hundred out of 110 patients had *ascites* of which forty three had *haemorrhage* in addition. Many had *peripheral oedema*. Of the remaining ten seven presented with haemorrhage as their major complaint which was associated with *jaundice* persistent anaemia or diabetes. Two had severe *anaemia* alone and in one the advanced degree of liver damage was asymptomatic and discovered accidentally at an abdominal operation. This last patient died of his first haemorrhage. Two patients in this group had right hydrothorax complicating and relieving the ascites similar in many ways to the patient described by Emerson and Davies. In both the hydrothorax and ascites improved *part passu*

Coma

Coma was a common concomitant of haemorrhage in this group. Its initial symptoms are important to recognise—the rather vacant politely unco-operative worried and unsmiling patient who often shows a peculiar flapping

tremor when the hands are held out horizontally (Adams and Foley, 1949). It will also be considered under the heading of 'Complications of Porta-caval Anastomosis'. Should the coma develop while the patient is under intensive medical therapy without predisposing cause, it is probably, but not certainly, an indication that the condition is beyond surgery. If it supervenes on a haemorrhage the chances of recovery are greater provided the haemorrhage can be stopped.

Much valuable work has been published recently on this difficult and troublesome subject (Latner, 1950, Farquhar *et al* 1950, Walshe, 1951, Sherlock, Summerskill, White, Phear, and Dawson, McDermott Wareham, and Riddell, Mann, Bollman *et al*).

DISCUSSION OF SYMPTOMS

1 Haematemesis and Melaena. In relating gastro-intestinal haemorrhage to portal hypertension it must be remembered that the commonest cause of haematemesis and melaena is undoubtedly peptic ulceration, so that a constant look-out needs to be kept for the symptom or sign that the bleeding is not due to an ulcer. An enlargement of the liver, a palpable spleen, and the stigmata of cirrhosis are the most important of such findings. In portal hypertension symptoms of dyspepsia may have been present, but very rarely is there any pain unless there is an associated ulcer. After the first haemorrhage, however, the patients may be able to appreciate a prodromal discomfort which will warn them that haemorrhage is going to occur within a few hours.

In children, however, haemorrhage often accompanies an acute specific fever, an attack of tonsillitis or a catarrhal infection of the upper respiratory passages. In one child (Case No. 63) a very severe haemorrhage was caused by chicken pox, with the rash extending right down the oesophagus. The parents of these children know more or less when to expect haemorrhages and have been able to mitigate the distress and shock. Chronically inflamed tonsils should be removed and particular care of these children should be taken during the course of any epidemic infection.

Haemorrhages usually occur at intervals of some months, but they tend to become more profuse and more frequent and often continuous. The bleeding usually comes from the oesophagus but may come from the stomach. The appearance of the vomited blood is rarely of diagnostic value, though haematemesis occurring from an oesophageal varix is more likely to be bright red and to contain clots than to consist of altered blood. Usually it is followed by melaena, occasionally there is melaena alone, and more rarely still the bleeding is a seep or diapedesis revealed by examination for occult blood in the stools.

A barium swallow X ray may be done as an emergency

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2 Hypersplenism and Splenic Anaemia Temporary portal decompression by haemorrhage will often cause a congested spleen to shrink and its presence may not become evident until some days after the bleeding has stopped. However splenomegaly can often be detected as an area of relative dullness to deep percussion over the lower ribs in the left mid axillary line. The blood count often shows leukopenia or thrombocytopenia.



FIG 60

Ca = No 114 Spider naevi on the chest of a man on the ten h day after porta caval anastomosis. All but the largest faded spontaneously.

3 Ascites In moderate and advanced cirrhosis the liver and also the spleen may often be obscured by the presence of ascites but the fluid itself is evidence of portal hypertension. The spleen and liver should be examined immediately after paracentesis. Small quantities of ascitic fluid are best detected by percussion around the umbilicus in the knee-elbow position.

4 The Stigmata of Cirrhosis On many occasions the presence of a deranged liver may be detected by noticing the apparently trivial superficial

changes which occur as a result of cirrhosis. The smallest blemish on the skin may be the clue to the underlying disease and lead a surgeon to hold



FIG 61A

Case No 138 Large spider naevus (advanced cirrhosis)

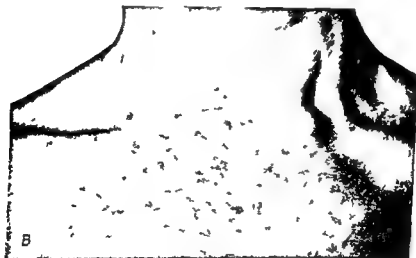


FIG 61B

Case No 223 Spider naevi in a patient with moderate cirrhosis

is hand correctly with a case of severe gastro-intestinal haemorrhage. The very triviality of these signs makes honest demonstration a difficult matter, because exceptional examples have to be taken. For instance, Fig 60 shows spider naevi covering the whole chest of a young man, almost like an exan

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them and Fig 61A again is an extreme example of a spider naevus. In this photograph of the lower half of a woman's face one can detect the sallow



FIG 61c

Case No 95 Spider naevi on the hand and forearm of a young girl



FIG 61b

Case No 164 Naevus of condyle on sometimes referred to as spider's legs

complexion and thin leathery patchy skin which is so characteristic of cirrhosis and which gives the patients an appearance of being older than they really are. Even in Fig 61b the naevi are more obvious than usual. In Fig

PORTAL HYPERTENSION

61b the naevoid condition appears more like wool dust scattered on the white skin, though it has been referred to as 'spider's legs'

Examination of the hands is often very revealing. In Fig 61c there are many small naevi present, and it is quite common to find the spiders on the palms or on the fingers. The man illustrated in Fig 62 was a mild cirrhotic without any evidence of liver damage as judged by biochemical tests, yet his palms were so strikingly blushed over the eminences that a confident diagnosis could be made.



FIG 62

Case No 70 Palmar blush (moderate cirrhosis)

The finger nails may be white (Terry, 1954). This condition is shown in the male (Fig 63, also showing clubbing), and in the female (Fig 64). The nails have the appearance of having been varnished with a matt opalescent varnish and are often curved. Pressure on the pulp of a normal finger will cause the colours in the nail bed to be accentuated. With the white nails of cirrhosis the pallor alone is accentuated. This point is well shown in Fig 64 where the index finger of the physician in charge has been photographed as a control.

An attempt should always be made to detect foetor hepaticus. Its presence does not necessarily signify an advanced degree of hepatic derangement, though it is more commonly found in patients who are about to go into coma.

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than in those who have a mild degree of cirrhosis. Two patients in the series lead perfectly normal lives but their entry into the examination room of the

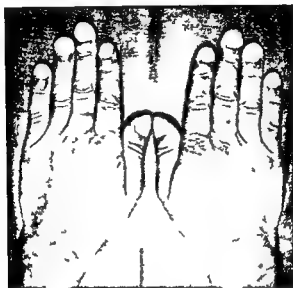


FIG 63

Case No 78 White nails and clubbed fingers

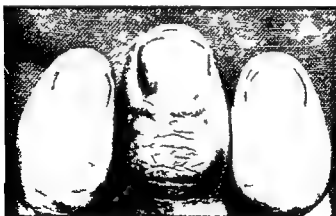


FIG 64

Case No 164 White nails compared with the normal

out patient department can sometimes be detected by the nose alone. It has a sweetish smell rather like that of stale shrimps.

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Other superficial changes associated with chronic liver disease and portal hypertension are illustrated in Fig 65, which shows gynaecomastia, recession of the suprapubic hair from the umbilicus, and dilatation of the superficial



FIG 65

Case No 167 To show ascites dilated veins recession of suprapubic hair and gynaecomastia and umbilical hernia

veins of the abdomen If there is marked flow from portal vein to the abdominal wall through an unobliterated umbilical vein a hum may be heard on auscultation A caput medusae is a most inconstant concomitant of portal hypertension as shown in considering the peripheral communications of the umbilical vein

THE INDICATIONS FOR OPERATION

1 Haemorrhage.

Haemorrhage can be catastrophic even in patients whose livers function normally In the cirrhotic patient there is further risk from liver failure or

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coma Fifty five years ago Langdon Brown showed that haematemesis occurred in thirteen out of forty-one patients with portal pylethrombosis and was fatal in eight. He refers to death being 'the ordinary termination'. Ratnoff and Patek, discussing cirrhosis in 1942, give a death rate of 42 per cent from the first haemorrhage, with only 30 per cent of all cirrhotics in their series alive at the end of the first year. Nachlas *et al* suggest that the figure should be higher, 60 per cent dying from the first bleed and only two thirds of the survivors alive at the end of one year. Higgins in 1947 analysed 115 cases of oesophageal varices and showed that 80 per cent were in cirrhotics and that the risk of death was one in four with each haemorrhage. Linton and Ellis showed a death rate of 50 per cent in the first year following recovery from haemorrhage. Sullens *et al* showed that cirrhosis was present in only 9.8 per cent of 305 patients with alimentary haemorrhage, but 63 per cent of these died in one year. In the present series it has been distressing to note the number of patients with moderate or severe cirrhosis who have died of their first bleed while under medical supervision. Some of them have not even had demonstrable varices. The conclusion is that if haemorrhage can be prevented by a safe surgical means it should be done. This cannot be questioned (Blakemore, 1951, Linton, 1951).

On the other hand there are occasional individuals e.g. Case No 62, who survive for many years with very little that could be termed abnormal. It is often striking how the initial haemorrhage in an adult reveals a fully developed state which may have been present since childhood. Reynell (1951) records that twelve patients with extrahepatic obstruction between them survived fifty haemorrhages without a single death. However, that some individuals can survive without an operation for many years does not infer that operation is unnecessary for the majority.

2 Ascites

All early attempts at the surgical treatment of portal hypertension were directed at the relief of ascites (e.g. Talma 1909) and a number of temporary successes was reported. To-day the pendulum has swung the other way and the tendency is to regard ascites as a condition which should never be treated by surgical means but which is the province of the physicians alone. There is no doubt that in circumstances of hypoalbuminaemia, fluid and salt retention and retention of antidiuretic substances such as aldosterone surgical decompression is not indicated (Popper and Elias 1955, Volwiler *et al*, 1950, Gray 1951). Correct dietary and medical treatment will in time bring about the absorption of ascitic fluid. With a minority, however, the liver function improves satisfactorily, but gross ascites remains intractable. It is these who are benefited by surgical treatment (Figs 28 and 80) (Habif *et al*, 1953).

When ascites complicates haemorrhage it becomes progressively worse after each bleed until it is permanently established (Case No 132). It is often necessary in these cases to take risks and to act boldly, within reason, to prevent another haemorrhage.

3 Splenic Anaemia.

The removal of the spleen for the relief of congestive splenomegaly and splenic anaemia is an obvious step and has been done a great number of times in the past. Until recently it was the operation of choice for Banti's syndrome (Banti, Lahey and Cyr). Evidence has already been adduced to show that it can be so occasionally, but to-day it is recognised that splenectomy usually fails to benefit the portal hypertension itself, although there are a number of patients, e.g., Case No 110, who are so ill from extreme anaemia that splenectomy must be done as the first step in the treatment, portal decompression coming later (Rousselet, Stock). In other circumstances porta caval anastomosis will produce an improvement in the blood picture.

4 Jaundice.

Jaundice is commonly and correctly regarded as a symptom of liver failure when associated with cirrhosis hepatis, and therefore a contra indication to surgical intervention. But there are patients in whom it is impossible to tell whether the jaundice is obstructive or 'hepatic' in origin and in them operative intervention is necessary to elucidate the true state of affairs. Jaundiced patients with portal hypertension are very liable to suffer from haemorrhages and, provided the liver functions reasonably well, it is logical to attempt porta-caval anastomosis to prevent bleeding. Patient No 246 (Fig 28) suffered from jaundice, haemorrhage, and ascites. After prolonged medical treatment she was regarded as a hopeless case. Eventually the liver improved enough for operative intervention to be considered feasible, the opportunity was taken, a porta-caval anastomosis was constructed and she made an excellent recovery. She is an illustration of what can be done in certain circumstances, she should not be taken as an illustration of what should be done for the majority.

5 Oesophageal Varices without Haemorrhage.

Some patients are discovered to be suffering from portal hypertension because of the splenic anaemia, splenomegaly, jaundice, or general ill health. They have not bled, though barium X ray or oesophagoscopy shows that they have oesophageal varices. Should they be treated prophylactically for haemorrhage? Linton in 1951, came to the conclusion that they should not, and most surgeons consider this the correct and safe attitude to adopt. Jahnke *et al* (1954) were of the opposite opinion. In my experience the longer that these patients remain under observation the greater is the number that have died of haemorrhage and some even when oesophageal varices have not

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been demonstrated. The more advanced cases of cirrhosis suffer the most. This suggests that, in cases of moderate cirrhosis, a shunt operation should probably be done as a prophylactic measure at the time when the patient is best able to tolerate surgical intervention. Case No. 118, seriously ill with ascites, did remarkably well under medical treatment and recovered good health. Large oesophageal varices were demonstrated and it was realised that should a haemorrhage occur it would be followed by serious deterioration of the liver and probably death. Porta-caval anastomosis was therefore done and he is now well and working three and a half years later.

In patients with extrahepatic obstruction or with mild cirrhosis it is probably better to await events. In these groups there is little risk if the patient is forewarned and the doctor prepared. It may be years before a haemorrhage occurs and operation becomes imperative.

CHAPTER IX

THE INVESTIGATION OF A CASE OF PORTAL HYPERTENSION

ENOUGH has been said in discussing and illustrating the gross pathology, symptomatology, physical signs, and indications for surgery to make it unnecessary to emphasise that a careful investigation is essential. It is assumed that a searching inquiry will be made into antecedent accidents, operations, or infections which might have caused damage to the portal vein, and into the past history with reference to the causes of cirrhosis such as infective hepatitis, dietary deficiencies, the habitual consumption of alcohol, the taking of medicines containing hepato toxic drugs, etc.

RADIOLOGICAL INVESTIGATIONS

1 Barium Swallow and Meal.

The demonstration of oesophageal and gastric varices by the use of the barium swallow is diagnostic. The meal should be completed in order to exclude an associated peptic ulcer or other cause of haemorrhage. The barium swallow is an examination which requires considerable skill on the part of the radiologist. When used as an emergency the patient need not be moved from his bed.

2 Radiographic Demonstration of Portal and Splenic Vein.

Portal venography has come to be regarded as an essential investigation in cases of portal hypertension. It was originally described by Blakemore and Lord in their article of 1945 in which the first successful series of porta-caval anastomoses was reported. The method used was the direct injection of contrast medium into a radicle of the portal vein on the operating table and the taking of an X ray photograph on a film placed in a cassette tunnel under the patient. This is still the simplest and the best method of obtaining an accurate picture of the portal vein itself, and this is the information that the surgeon requires. Many valuable papers have been written concerning this method, notably by Santy and Marion (1953), and Leger *et al* (1955) in France, de Sousa Pereira *et al* (1949) in Portugal, and du Boulay and Green (1954) in England. It has been used by the author since the beginning of 1949 supplemented by the alternative method of splenic venography in recent years. My purpose has been to eliminate error in visualising the portal tree. The direct method has the disadvantage of not being available until the abdomen has been opened.

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Percutaneous transsplenic venography was introduced in 1951 by two independent groups of workers Abeatici and Campi in Italy, and Dreyer and Budtz Olsen in South Africa (Bahnsen, Sloan, and Blalock had obtained splenic venograms accidentally in 1942 when injecting an epigastric sinus

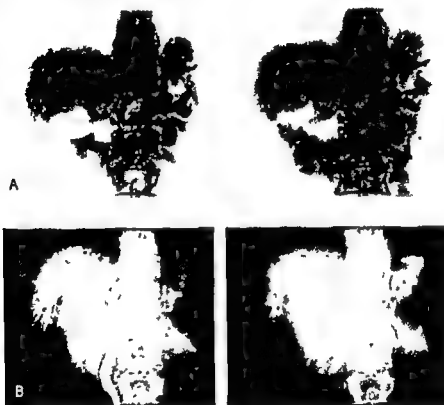


FIG 66

Case No 235 Comparable splenic (A) and portal (B) venograms obtained at the same operation on a case of moderate cirrhosis. In A the portal vein is no more than outlined laminar flow giving the appearance of a large central thrombus. In B on the other hand the portal vein is well filled (The diiodone from the splenic venogram appears as a pyclogram in B)

with iodochlorol). The contrast medium is injected directly into the pulp of the spleen. It passes immediately into the portal circulation, delineates the splenic vein, many of the collateral anastomotic channels, and often the portal vein itself. The development and popularisation of this method has been of the first importance in emphasising the value of radiological demonstration of the portal tree (Walker, Middlemiss, and Nanson, Dogliotti, Abeatici, and

Campi, Atkinson, Barnett, Sherlock, and Steiner) It can be done under a local anaesthetic and is therefore available as a diagnostic measure This evidence may assist in deciding which operation is the most suitable, and therefore in the placing of the incision, provided due cognisance is taken of errors in the visualisation of the portal tree For example, splenic blood may be diverted up anastomotic vessels so that little or none reaches the portal vein (Fig 66A), and laminar flow or streamlining may give a picture suggestive of partial obliteration by a thrombus These disadvantages are of little account if they are recognised and confirmatory evidence is obtained by direct portal venography after the abdomen has been opened (Fig 66B) This combination of the two methods is without doubt the best approach (Rousselot, Ruzicka, and Doehner, 1953, du Boulay, Green, and Hunt, 1957)

Splenic puncture venography suffers the additional disadvantage that it is not an absolutely safe procedure (Gvozdanovic *et al*, Reynolds, Mikkelsen *et al*, Figley *et al*) Cases have been recorded of damage having been done to the splenic capsule with considerable resultant perisplenic haemorrhage amounting to litres and occasionally causing death In my experience the amount of blood which leaks from the spleen following splenic venography has been rarely less than 40 ml, as found at operation immediately after forty such investigations, and on one occasion was as much as 600 ml The method should never be used where the clotting power of the blood is diminished either by a marked reduction of platelets or prothrombin It is a difficult method when the spleen is not considerably enlarged and of course, it is out of the question whenever the spleen has previously been removed In the present series there were twenty cases of 'post splenectomy bleeders' in which the only possible method of obtaining the portal venograms was by direct portal venography

Photographs obtained pre-operatively by splenic injection venography are taken on the static machine and are usually of better definition than those obtained with a mobile apparatus on the operating table However, provided the technique of the team is good the photographs obtained with a 'portable' will provide the information that is required, as shown by the illustrations of the present essay

Patients with portal hypertension are ill distressed and depressed They have usually been submitted to a great number of investigations before the surgeon sees them Their morale is often low from repeated haematemeses or ascites For these reasons I believe in sparing them as many needle pricks and special tests as possible, reserving splenic venography until they are under the general anaesthetic It is the kinder method and just as efficient

INVESTIGATION OF A CASE OF PORTAL HYPERTENSION

3 Other Investigations

X RAY (a) *Chest* An X ray examination of the chest is of value even in patients who have no pulmonary disease. Operations for portal hypertension may involve one or other dome of the diaphragm. A preliminary picture is necessary for comparison with X rays obtained during the post operative phase.

(b) *Intravenous Pyelogram* This again is a necessary investigation if there is any question that a lino renal anastomosis may be done. It is not always possible to be sure that the left kidney will be preserved and before sacrificing it it is essential to demonstrate that the right kidney is functioning normally.

PATHOLOGICAL INVESTIGATIONS

1 Haematological

Complete examination of the cellular elements of the blood is absolutely necessary. The reticulocytes should be counted and a sternal puncture done if there is any question of failure of haemopoiesis. Transfusions may be required so that the blood group should always be ascertained early in the investigation. When the liver is diseased it is important to estimate the prothrombin content of the blood and its response to treatment with massive doses of vitamin K. It is wise to exclude the possibility of syphilis in every case by routine serological investigation (*vide* Case No. 205).

2 Liver Function Tests

A great number of liver function tests have been devised and reference should be made to books such as those by Lichtman and by Sherlock and articles such as Rennie's. The tests serve two functions—diagnostic and prognostic—which are not necessarily the same. For example the flocculation tests, the electrophoretic pattern of the serum proteins and the urinary urobilin may give an indication of cirrhosis at a time when estimation of the serum albumin gives a normal result. Yet the latter test is of far greater value in assessing the patient's prospects.

Serum albumin is made in the liver and the quantity present is the most reliable index of liver function. The critical level below which operative intervention will not be tolerated is 3.2 gm / 100 ml.

Pseudocholinesterase, the non specific cholinesterase found in the serum, is another substance formed in the liver which gives an indication of liver function by the quantity present (Vorhaus *et al.* Wilson *et al.* and Lehmann). The normal range varies considerably (55–120 units) so that it is not so reliable as an absolute index as the serum albumin. It has sometimes been useful diagnostically especially in children and in cases of jaundice where the

common tests can be misleading. It has also been of value in eliminating cases in which the liver was not cirrhotic. For example, a man with ascites had a total serum protein of 4.4, albumin 2.1, globulin 2.3, and a pseudo cholinesterase of 122 units. The latter finding ruled out cirrhosis and suggested the diagnosis of wet beri beri. Appropriate treatment was promptly instituted and subsequent progress confirmed that the liver was normal.

It is in the changing level that the pseudocholinesterase is of most value. An advanced cirrhotic on 6th November had serum proteins of 2.5 albumin/4.2 globulin and a pseudocholinesterase of 50 units. On 9th December the albumin had risen to 2.9, yet the pseudocholinesterase had fallen to 27 units. Clinically she appeared to be holding her own, but she passed into coma and died on 1st January, showing that the fall in pseudocholinesterase was the true pointer to her fate. Similar cases had falls of 47 to 28 and 27 to 18 units before developing coma.

The pseudocholinesterase should be estimated pre-operatively whenever a patient is to be given scoline as a relaxant. Patients with low levels are in danger of prolonged apnoea (Evans *et al.*, 1952 and 1953, Calvert *et al.* 1954). This vital matter and the prognostic value of the test suggest that it should be more frequently used.

The bromsulphthalein elimination test is another investigation of value (Blakemore, 1949) and it is a fair reflection of the detoxicating capacity of the liver.

The serum bilirubin and alkaline phosphatase have been estimated in every case of cirrhosis. The knowledge is essential in the presence of jaundice and is useful as a baseline in all cases. Icterus can be a complication of operative treatment and it is necessary to know the pre-operative levels. Norcross *et al.* consider that it is not possible to correlate structural change and functional abnormality except in relation to biliary retention.

3 Other Special Investigations.

Various other investigations may be considered advisable from time to time. For example, if there is question in the diagnosis between cirrhosis hepatis and primary carcinoma of the liver, a puncture biopsy of the liver or peritoneoscopy may help. Oesophagoscopy and even gastroscopy may be necessary when radiological methods have failed to demonstrate an abnormality in the oesophagus or stomach, though instrumental investigation should be avoided if possible on account of the slight risk of haemorrhage.

Aspirated ascitic fluid must always be examined. It is useful to know the protein loss. Too much reliance must not be placed on the finding of malignant looking cells, because great experience is needed to differentiate between them and degenerate mesodermal cells commonly found. In Patient

INVESTIGATION OF A CASE OF PORTAL HYPERTENSION

No 205 therapy was wrongly applied because of a mistaken diagnosis of malignancy based on these free cells

There are numerous ingenious methods of estimating the portal pressure by splenic puncture, catheterisation of the hepatic veins (Paton, Reynolds, and Sherlock, 1953) puncture of oesophageal varices (Allison, 1951), and liver puncture (Reimann *et al* 1955) The speed with which blood passes through the portal system can be approximately estimated by the injection or instillation of substances into various parts of the alimentary canal Newman and Cohen (1949), Arafa *et al* (1954), and Giges and Teschan (1952) used ether vapour per rectum with the nose as receptor, Deterling *et al* (1955) used radioactive sodium instilled into the prepared colon with a scintillation counter over the brachial artery as detector, Henning *et al* (1950) used acetylene, Akita *et al* (1956) bile salts The surgeon is in a fortunate position in that he can discover all that is necessary in these respects at the time of the operation Such examinations may, however, be of value in testing the patency of a venous shunt, but the only valid test of patency is that a shunt shall deflate oesophageal varices and stop haemorrhage, or lead to the disappearance of ascites

CHAPTER X

THE PREPARATION OF PATIENTS FOR OPERATION AND THE TREATMENT OF CIRRHOSIS

1 Extrahepatic Obstruction and Mild Cirrhosis

THESE patients require the same preparation for operative intervention as in any other disease where a major operation is to be undertaken. They are built up for a matter of two or three weeks following a haemorrhage. Once they are well again, operation should not be unduly delayed. If they live far away they should not be allowed home until after radical surgical treatment has been completed, because of the risks of another haemorrhage.

2 Moderate Cirrhosis.

Preparation is by medical measures until their liver function has been built up to the best that can be achieved. It may be as much as two months but seldom longer.

3 Advanced Cirrhosis.

These patients require intensive and prolonged medical treatment with all the skill and encouragement that can be mustered. It is all that can be done for more than half the patients with advanced cirrhosis. They have not had a haemorrhage and operation of any sort is quite unjustified. To emphasise points that are important in the medical treatment is not out of place (see Patek *et al*, 1948, Cullinan).

THE TREATMENT OF CIRRHOSIS

High Protein Diet.

The animal protein intake per day should be 120 grams. Some, particularly the younger patients, are able to consume more than this, others not as much on account of the stupor that excessive ingestion of protein induces. The protein should be first class in quality and attractively served. It is always worth helping patients who can manage this treatment at home by instructing them and their wives or whoever is responsible for looking after them, in the protein contents of the different foodstuffs and how best they may be prepared in a palatable manner. The publication of the Medical Research Council on the "Chemical Composition of Foods" (McCance and Widdowson 1954) is a good book of reference and enables grams consumed per day to be translated into pounds and ounces, so that the layman may be told how

PREPARATION OF PATIENTS FOR OPERATION

much meat and milk, fish, eggs and cheese he requires to buy from the shop in order to provide the necessary daily protein intake

Meat should be lean and of good quality, fish white and not oily, grilling and roasting are the best methods of cooking, with steaming and light stewing allowed. Garnishing may be done simply and tastefully with parsley butter and other herbs

Protein supplements such as Casilan can be added to soups, gravies, puddings, and ice-creams. White of egg is another useful adjunct

It must be remembered that the advanced cirrhotic with peripheral oedema probably does not absorb as much from the alimentary canal as the normal patient. The intestines are thickened, sodden, and sometimes matted with oedema and lymph

Fresh fruit and vegetables in good quantities should always be eaten to supply vitamins in a natural form. Some can be prepared so that a great deal can be consumed without the patient being surfeited. For example, 1 lb of tomatoes rubbed through the sieve provides a reasonable mid morning drink, but a mountain eaten raw. Four oranges as juice to drink is not excessive

Carbohydrates Fats Flavourings etc—Bread butter, cream, jam, honey, marmalade, are all allowed. Barley sugar, boiled sweets, and glucose are preferable to chocolate, nuts and marzipan. Potatoes and all pasta are of value but should not be eaten in so great a quantity as to reduce the appetite for the more essential proteins and vitamins. This also applies to heavy and fatty foods such as rich stews, goulashes and Welsh rarebit. These and all cooking fats, margarine, pastries, and heavy cakes are tolerated badly by the cirrhotic and should not be given. Heavy frying is bad

It is self-evident that alcohol is absolutely forbidden. But the alcoholic cirrhotic is no more easy to cure of his vice than any other and many will go to endless lengths to get a drink

Fluid restriction—Any cirrhotic with a tendency to ascites should if possible be restricted to two and a half pints of fluid a day

Salt Restriction—All foods should be salt free, with special butter, bread and porridge. No salt should be added in the cooking or at table. Salt substitutes may be tried but they are not usually necessary. Most cirrhotics accept this restriction and prefer their food so. One patient even complained that 'a meal out tasted like brine'

Diuresis may be induced for the elimination of fluids by the use of mercurial diuretics though ammonium chloride is sometimes badly tolerated

Specific Therapy.

If the cirrhosis is of a specific type—e.g. syphilis arsenic—specific measures are adopted (Nicol, Nicol and Terry, Wade and Frazer)

Special symptomatic treatment may also be advisable, e.g. methyl testosterone for the itch of jaundice (Lloyd Thomas and Sherlock)

In General.

The patients should be kept at rest but not completely restricted to their beds unless there is an imminent risk of haemorrhage. Paracentesis abdominis should not be deferred unduly long and should be done with every care to avoid upsetting the patient. If ascitic fluid is allowed to collect excessively it will cause basal pulmonary collapse, pneumonia, and interference with the heart's action, leading to an anoxic condition which must in all circumstances be avoided in patients with cirrhosis hepatis (Fig. 67)

The Treatment of Coma.

Should coma supervene, proteins should be cut from the diet immediately. Carbohydrate, particularly glucose, and vitamins should constitute the bulk of the food. Vitamin concentrates given parenterally are of great value. Tetracycline is given to reduce those bacteria of the intestine which produce ammoniacal substances toxic to the brain (Farquhar *et al.*, Phear *et al.*, following Markowitz *et al.*, 1949). If the patient is unable to swallow and is being treated parenterally for haemorrhage, glucose in 20 per cent solution should be given through a polythene tube threaded up into one of the main veins of the body. Every effort should be made to evacuate protein containing residues from the bowel. Opiates and barbiturates are forbidden. A clear airway is maintained.

Walshe (1953) suggested the use of sodium glutamate, either intravenously or by mouth. Sometimes it appears to produce an improvement and should always be given a trial.

A method temporarily successful on one occasion in restoring consciousness was to give a replacement transfusion of two pints of fresh blood.

Dialysis by means of the artificial kidney might also be worth a trial.

Coma can be precipitated by mesenteric thrombosis, with clot extending up into the portal vein and liver, or intrahepatic thrombosis occurring post-operatively. In these circumstances benefit should be obtained from treatment with heparin, given cautiously because of the risk of haemorrhage. Such treatment was indeed accompanied by rapid recovery from coma in two patients who had evidence of portal vein thrombosis at operation, but whether the heparin or the glucose was responsible it is impossible to say (Hunt and Whittard 1954).

As a precaution against the development of coma following porta-caval anastomosis, it is wise to give patients with moderate and severe cirrhosis a protein free diet for twenty four hours before such operations and to ensure

PREPARATION OF PATIENTS FOR OPERATION



FIG. 67

Case No. 42 X rays of chest of patient before and two days after paracentesis abdominis. Pulmonary collapse and pneumonia relieved within forty-eight hours.

PORTAL HYPERTENSION

that the bowel is clear of residue by giving a colonic washout on the morning of the operation. Opiates are not given post operatively.

If coma follows haemorrhage, effective control of the haemorrhage is the first and most important step in the treatment of the coma.

SELECTION OF CASES OF ADVANCED CIRRHOSIS FOR OPERATION

Intensive medical therapy is required in cases of advanced cirrhosis for at least three months before seriously considering operative treatment for haemorrhage or intractable ascites. It is useful to have the patient's progress

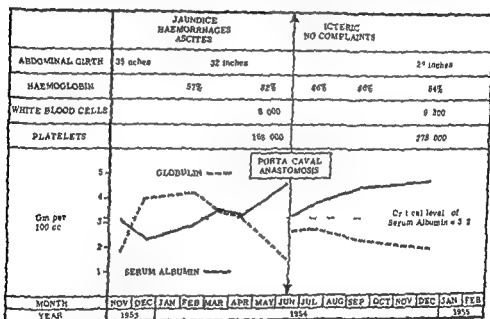


FIG 68

Case No 149 The progress of a case of congenital cirrhosis over sixteen months from a state of distressing ascites, recurrent haemorrhages and jaundice to normal boyhood and school life.

and certain critical liver function tests set out on a graph for easy study and assessment (Fig 68). The degree of ascites can be recorded in two ways (1) as daily chart of the abdominal girth and (2) of the quantity of ascitic fluid formed (the quantity withdrawn at paracentesis divided by the interval in days between tappings) (Fig 69). Tests of the haemoglobin are done frequently and the serum albumin is estimated every fortnight. It must rise to above 3.2 gm/100 ml (Blakemore 1949) before operation can be contemplated. Other liver function and blood tests are done only as they are required. It has already been emphasised that such patients do not appreciate over

PREPARATION OF PATIENTS FOR OPERATION

investigation and it is my firm conviction that they require encouragement rather than venepuncture, let alone other more penetrating types of puncture.

A bad prognosis is indicated by a rise in the serum bilirubin and alkaline phosphatase and a lowering of the serum albumin and pseudo-cholinesterase and of the plasma prothrombin.

Clinical assessment must always carry as much weight as all the laboratory investigations.

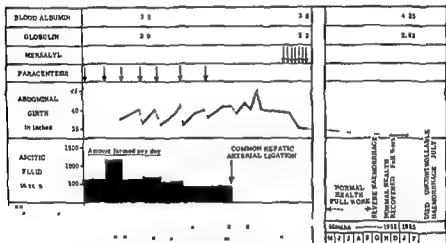


Fig. 69

Case No 61 The progress chart of a case of advanced cirrhosis for whom the common hepatic artery was ligatured

A hopeful attitude may be adopted with the young and with the resilient. For instance, it is in a patient's favour if he has survived numerous haemorrhages in the past and the quicker he can pull up the better.

In conclusion, absolute criteria of operability are impossible to define. When in doubt, persist with medical treatment and re-assess in one month unless haemorrhage threatens. Haemorrhage is the one symptom and indication that may force the surgeon's hand. He must decide which is the greater risk, death following operation with reasonable prospect of a good life if the patient recovers, or, without operation, little chance of anything better than precarious health, with probable death from the next haemorrhage.

CHAPTER XI

THE EMERGENCY TREATMENT OF HAEMORRHAGE

THE emergency treatment of haemorrhage and the restoration of the haemoglobin level to as near normal as possible is the most important part of the build up for definitive surgery in portal hypertension. For the purpose of the present discussion it is assumed that the diagnosis has been established.

1 Transfusions.

The bleeding patient is treated first in the simplest manner. He is put to bed, an intravenous drip is set up, and he is given sedatives. Paraldehyde is the safest. Cases with extrahepatic obstruction or mild cirrhosis will tolerate

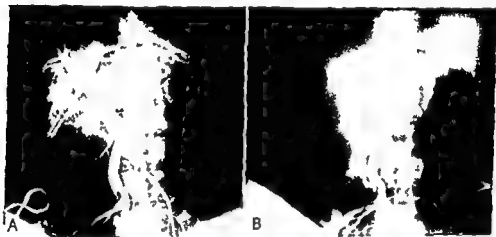


FIG. 70

Case No. 179. Emergency and definitive treatment of haemorrhage illustrated by portal venograms taken collapsed in position (A) and after tamponade (B). Ten

opiates and barbiturates well. With moderate cirrhosis these should be given sparingly, and with advanced cirrhosis not at all. Transfusions are prepared and are given soon rather than late in order to keep pace with the blood loss. In the majority of patients this treatment will be successful and the bleeding will stop.

2 Compression of the Lower Oesophageal Varices.

Should the simple measures fail tamponade by means of a Sengstaken Blakemore (1950) tube should be resorted to (Fig. 70). Barnett and Cohen

EMERGENCY TREATMENT OF HAEMORRHAGE

(1949), and Boerema (1954) also utilised similar methods with success. This causes considerable distress to the patient. The tube itself is large and has to be passed through the mouth, where it is very uncomfortable. When the balloon is blown up, spasm of the oesophagus and cardia occurs with retro-sternal, thoracic, and epigastric pain. However, the patients, aware of their desperate circumstances, will put up with it. The balloon must not be left blown up for more than thirty-six to forty-eight hours otherwise it in its turn will induce secondary ulceration, which will produce further haemorrhage. As soon as the bleeding is thought to be under control, the balloon should be deflated and the tube removed gently a few hours later. If the bleeding recurs, the tube should be returned and the patient prepared for the next stage in the treatment which, though operative, will be considered briefly here. Linton and Ellis (1956) give an excellent description of the emergency and definitive treatment of haemorrhage.

3 Emergency Operative Treatment.

(1) The safest and simplest effective operation, albeit a major and difficult one, is that of *oesophagotomy and ligation of the oesophageal varices* done by Boerema in 1949 and described by George Crile jun., in 1953 (and see Appendix 4, B). Its position in the treatment and prevention of haemorrhage from oesophageal varices is to save life, and it is unfortunately of no more than temporary benefit.

(2) *Porta-caval Anastomosis*—This has been done sometimes as an emergency measure, even though it is an extremely risky procedure. It has only been attempted twice in the present series, in both patients (Cases 132 and 179) with temporary success. One died six months later, the other two years later, both of liver failure. O'Sullivan and Payne have done nine emergency porta-caval anastomoses with three operative deaths and three late deaths. These are poor figures, but better than allowing these unfortunate individuals to die.

(3) Lastly, *proximal gastric resection* (Appendix 4, A) has been done on two occasions as an emergency, both in cases of extrahepatic obstruction, once in the small hours of the morning to save the life of a little girl for whom every other operation had been done (Case No. 63), and on the other occasion for a man who had had twenty six pints of blood transfused and yet continued to bleed (Case No. 6). Both have done well, the girl at school and the man at heavy work for seven and a half years.

The Place of Partial Gastrectomy for Associated Peptic Ulcer.

A special difficulty is sometimes encountered when a patient is admitted with acute haematemesis following a history typical of peptic ulceration. The

haemorrhage becomes severe and cannot be controlled by medical treatment. An exploratory operation is done with a view to resecting the stomach. An ulcer and a cirrhotic liver are both discovered. What should the surgeon do? Should he treat the ulcer by resection or should he close the abdomen and allow time for medical treatment of the cirrhosis hepatis, risking immediate death from haemorrhage? Very often such an emergency operation will be done in circumstances where the full facilities for the investigation of a case of portal hypertension are not available. However it can be stated emphatically that if the bleeding is coming from an eroded vessel in the base of a duodenal or gastric ulcer resection as an emergency is essential. It should of course be done as expeditiously as possible. Lipp and Lipsitz stress this necessity for operation. The portal hypertension will have to be observed and treated according to developments. If the bleeding is not coming from the ulcer but from oesophageal varices partial gastrectomy must not be done especially when the cirrhosis is severe. Such cases are of extreme difficulty and must be dealt with as the occasion arises. Often gastrotomy will help in arriving at a decision. Such measures as excision of the ulcer alone must not be forgotten.

There are certain surgeons who advocate doing a blind gastrectomy for massive haematemesis when no definite ulcer can be found. Whatever the merits or demerits of this opinion may be when the liver is normal a blind gastric resection is absolutely contraindicated in the presence of a cirrhotic liver (Case No. 246).

CHAPTER XII

THE OPERATIVE TREATMENT OF PORTAL HYPERTENSION

✓ THE DEVELOPMENT OF MODERN METHODS

THE history of the modern operative treatment of portal hypertension begins with Eck who suggested in 1877 that his porta-caval anastomosis applied to human beings would provide the correct treatment for portal stasis. Of Eck's three dogs only one survived the operation and then ran away, so that there was no tangible proof of what had been achieved. During the years immediately following his brief article many survivors were obtained by other workers, proving that porta-caval anastomosis was possible in animals. Vidal in 1903 has the distinction of being the first surgeon to have done the operation on a human being with survival for four months. De Martel in 1910 and Rosenstein in 1912 each constructed a porta-caval anastomosis in-continuity without ligaturing the hepatic end of the portal vein, in the hope that some of the blood would continue to pass into the liver. MacIndoe in 1928 reiterated this suggestion, that the porta-caval anastomosis in-continuity should be the most satisfactory treatment of portal hypertension. Meanwhile many other minor shunt operations were being done, but no work of any practical significance came to the fore. In 1945, however, Blakemore and Lord, working in Whipple's clinic and following on the work that Rousselot had done during the preceding years, published the first series of portal to systemic venous anastomoses and demonstrated that the method was practicable in man. They joined the splenic to the renal vein end-to-end, or the portal vein to the vena cava end-to-side, using the Blakemore Lord tube Anastomosis of the vessels by the suture method, as developed by Blalock, soon followed. Linton *et al* (1947 & 1949) showed that it was unnecessary in the majority of cases to sacrifice the left kidney and made the lieno-renal anastomosis also by the end to-side method. Welch (1947) introduced the Tom Smith clamp (see Roxburgh) for isolating no more than a small segment of the inferior vena cava during the unhurried construction of the anastomosis, allowing blood to flow up the rest of the channel unimpeded. In this country Learmonth in Edinburgh and Milnes Walker in Bristol were the first to do porta-caval anastomoses in any numbers.

In these operations the surgeon made no claim to be able to arrest a progressive disease of the liver. He tried only to decompress the portal tree and so to prevent haemorrhage or to eliminate ascites. Success was achieved only when the liver could stand up to deprivation of its portal supply of

blood. An alternative approach was being developed to skirt this problem aiming to do no more than to stop haemorrhage by the elimination of the bleeding segment. Crafoord and Frenckner in 1939 injected the veins in the oesophagus through an oesophagoscope. Phemister and Humphreys in 1947 removed the lower end of the oesophagus and the proximal end of the stomach. Tanner (1950) modified this latter procedure by transecting the stomach just below the cardia through the abdomen after isolating the cardiac end of the stomach from as many vascular communications as possible. A complete list of operations up to 1947 is given by Baronofsky who with Wangenstein advocated extensive gastric resection for the reduction of gastric acidity and thereby the prevention of erosions.

Lastly Rienhoff in 1951 introduced the operation of hepatic arterial ligation as an alternative method of relieving portal congestion. This operation completes the list of those done for the patients under review with the exception of hepatic arterialisation (see page 108).

The operations and the results are considered in the groups into which the patients have been divided.

THE ANALYSIS OF OPERATIONS AND RESULTS

1. EXTRA HEPATIC PORTAL OBSTRUCTION

(a) Congenital Obliteration and Stricture (19 patients)

One died without operation (Case No. 21). Thirty nine major operations have been done on the other eighteen without an operative death. The mechanical problem is greater than in cirrhosis because these patients usually do not have a portal vein. Relief of haemorrhage has been attempted by one method after another as each has failed. Patient No. 63 illustrates the difficulties. This child had a lienorenal anastomosis using a poor splenic vein then a makeshift porta-caval followed by gastric transection then sclerosing injection of her oesophageal varices and finally transthoracic proximal gastric resection. She now has a mild erosive oesophagitis with occasional haemorrhages.

SPLENECTOMY Five patients had had their spleens removed and had continued to have dangerous haemorrhages before being referred for subsequent operative treatment. The sixth had splenic vein thrombosis at the time of operation so that nothing more than splenectomy was possible. The last patient died later of mesenteric thrombosis. The result of splenectomy alone in this group has thus been total failure. It should therefore not be done unless it is unavoidable as in one additional patient recently who had a simple splenectomy because no suitable venous channel was available for a shunt.

OPERATIVE TREATMENT OF PORTAL HYPERTENSION

LIENO-RENAL ANASTOMOSIS—Lieno renal anastomosis has been done ten times and seven have thrombosed. The reason for the bad results is that the splenic vein is small and poor in quality and seems to be possessed of the same tendency to obliteration as the portal vein. This tendency is accentuated when the portal pressure is reduced. There are thus only three successful results (30 per cent), but these patients are normal healthy children the longest survivor having had no haemorrhage or other symptom of disease for four and a half years.

End to-side anastomosis was done six times, of which four have thrombosed. End to-end anastomosis was done four times of which three have thrombosed, two were exceptionally difficult, one was done using a Blakemore-Lord tube. The fourth, a successful case had two renal veins and one was used for the shunt without removing the kidney (Case No. 219).

PORTA-CAVAL ANASTOMOSIS—Direct end to-side porta-caval anastomosis has been done three times, once for the patient with portal vein stricture (Fig. 11) whose lieno renal anastomosis had thrombosed, and on the two other occasions for the patients with Cruveilhier Baumgarten disease, one of whom had had previous splenectomy (Fig. 50) and the other previous lieno renal anastomosis (Fig. 14), in both of whom clot had to be scraped from the portal vein before the anastomosis could be made. All three are alive and well and have had no further haemorrhage or other symptom. It seems, therefore, that where a portal vein is present porta-caval anastomosis should be done in preference to lieno renal, even though it means sacrificing the contribution which a patent umbilical vein makes to portal decompression. Abdalla and Da Costa are of the opposite opinion.

MINOR ANASTOMOSES—‘Makeshift’ anastomoses (Linton, 1949) have been done on five patients who had no portal or splenic vein. Superior mesenteric, inferior mesenteric, and cavernomatous channels from the gastro-hepatic omentum have been used for these shunts and all have thrombosed. It seems that all such trivial attempts at anastomosis are foredoomed to failure, especially in children. One girl with a thrombosed makeshift shunt has had only one minor haemorrhage in the six years since operation.

Blakemore and Fitzpatrick have elaborated a technique for vein grafting between the stump of the splenic vein and the inferior vena cava in cases of ‘post splenectomy bleeders’. The shunt is heparinised by the local instillation of heparin through a fine polythene tube. The late results of this ingenious method have not yet been published.

GASTRIC TRANSECTION—This has been done five times. All the patients have bled again.

PROXIMAL GASTRIC RESECTION—This has been done nine times. Four have been successful for two, two and a half, four, and seven and a half years.

since operation, though one child developed a stricture at the site of the anastomosis which required treatment by dilatation. These patients though comparatively fit, are underweight and liable to a dyspepsia which can usually be controlled by mild dietary restriction and preprandial alkali. None is in such good physical condition as the patients with successful shunts. The suggestion has been made by Clatworthy that gastric replacement by interposition of a segment of jejunum might obviate the complications.

One patient is a failure on account of severe reflux peristalsis and regurgitant oesophagitis, which from time to time becomes very distressing and prevents her from eating a satisfying meal. Four patients have been failures on account of haemorrhage at thirteen, twenty nine, thirty six, and thirty-eight months after operation. The last has no demonstrable varices but the bleeding comes from an oesophageal erosion.

In Summary, operations on young children with congenital portal vein obliteration are disappointing. The first operation should be lieno renal anastomosis if there is a workable splenic vein, or, better still porta-caval if there is a good portal vein. (If there is no major venous channel, simple splenectomy may be unavoidable.) The second operation should be proximal gastric resection or some modification. There is also a place for sclerosing injections.

(b) Portal Vein Obliteration due to Thrombosis, etc.

Three patients, all adults in whom the thrombosis followed portal pylephlebitis, have been well since end to side lieno renal anastomosis. The average follow up is of nearly three years. All had good veins. Two patients with traumatic thrombosis have both been improved by proximal gastric resection following failure of splenectomy and lieno renal anastomosis respectively.

Other causes of extrahepatic compression and invasion have been dealt with as they have been found, e.g. splenectomy in Case No. 74 nephrectomy in Case No. 47.

2 MILD CIRRHOSIS (33 patients)

In six patients there has been no need for operative treatment. The cirrhosis and portal hypertension were discovered accidentally during the course of operative treatment for biliary obstruction carcinoma of the stomach duodenal ulcer, appendicitis, and hernia.

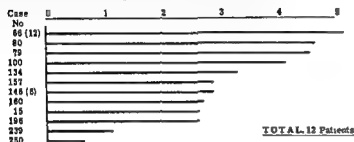
Porta caval and Lieno renal Anastomosis

The results of twenty three shunt operations done in twenty one patients are set out in the table (Fig. 71). There were twelve porta-caval and eleven lieno renal anastomoses. There has been no operative death and only one late death due to liver failure while the patient was convalescing in the country.

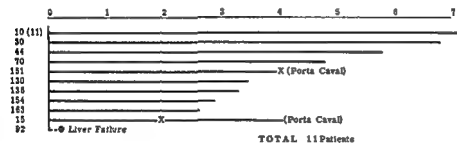
OPERATIVE TREATMENT OF PORTAL HYPERTENSION

Two end-to-side lieno-renal anastomoses have thrombosed and in both these patients (Nos 15 and 151) subsequent porta-caval anastomosis has been successful. Otherwise none has had further haemorrhage. The average follow up period for this group has been three years.

MILD CIRRHOSIS. TIMES OF SURVIVAL AND FOLLOW-UP IN YEARS FOLLOWING PORTA-CAVAL ANASTOMOSIS



AND FOLLOWING LIENO-RENAL ANASTOMOSIS



CAUSES OF DEATH WRITTEN IN

OPERATIVE MORTALITY = NIL
SUCCESS RATE = 87%
Average follow-up = 3 years

KEY -
—— = SURVIVOR
—•— = Late Death
—X— = Thrombosed

FIG 71

Eighteen out of the twenty-one patients are able to lead normal working lives a success rate of 86 per cent which cannot be questioned. The remaining two, women with postnecrotic cirrhosis, aged 52 and 46 at the time of their operations, have suffered from episodic stupor for three and two years respectively. Their livers are deteriorating slowly, but they have had no haemorrhage. By less rigorous standards or on a shorter follow up they would also be judged successful.

These results confirm the opinion of many surgeons—Blakemore, Linton and Ellis Welch, Milnes Walker—that a shunt operation is at present the operation of choice for the mildly cirrhotic patient with Banti's syndrome.

Splenectomy.

This was done on six occasions and two were successful. In one successful patient with splenic anaemia, Case No 249, it was possible to demonstrate satisfactory decompression of the portal tree at the end of the operation. In another it was done for splenic anaemia with haemorrhage, and the patient has been well since operation nearly three years ago. One patient with portal and splenic vein thrombosis died some months after operation from a pulmonary embolus. The other three continued to bleed until treated by some other operation, since when they have been well for periods of two and a half to five years. In one it was proximal gastric transection (Case No 165), in another a difficult porta-caval anastomosis (Case No 66), and in the third proximal gastric resection (Case No 155).

Gastric Transection and Resection (one of each, both successful)

In cirrhotics, transection and resection operations can be of exceptional difficulty. Patient No 165 had such dense adhesions of such thickness, brittleness, and vascularity that the stomach wall was found only after two hours of operating. Such operations should not be attempted as emergencies in cirrhotics and are less likely to be effective than in extrahepatic obstruction.

3 MODERATE CIRRHOSIS (75 patients)

No operation was done for twenty three. Of these, thirteen have suffered from ascites and are maintaining reasonable health with medical treatment. Seven have died, five of liver failure (one following his first haemorrhage), one of cerebral thrombosis, and one of renal failure. The remaining two have haemorrhages, one considered a bad operative risk by reason of age and the other under treatment by injection of varices, his portal and splenic veins being totally thrombosed.

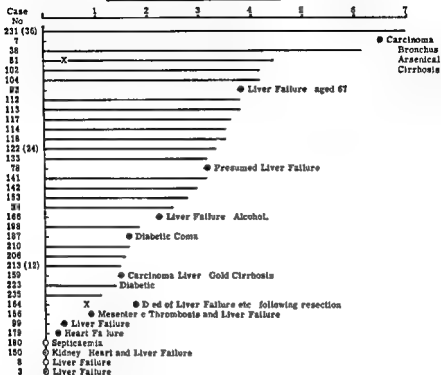
Porta caval and Leno renal Anastomosis.

Portal to systemic venous anastomosis was done for forty seven patients and the results are set out in the table (Fig 72). The operative mortality was 10.6 per cent and the success rate 70.2 per cent, the average period of follow up being nearly three years. Some patients who have died of other diseases have been considered as successes from the point of view of their portal hypertension, e.g., Patient No 7 (arsenical cirrhosis), who died of carcinoma of the bronchus six and a half years after porta-caval anastomosis for ascites, Patient No 187 who died of diabetic coma nearly two years after his anastomosis, and Patient No 159 (gold cirrhosis), who died of carcinoma of the liver, also two years after his operation. The porta-caval anastomosis in these three patients was shown postmortem to be satisfactory.

OPERATIVE TREATMENT OF PORTAL HYPERTENSION

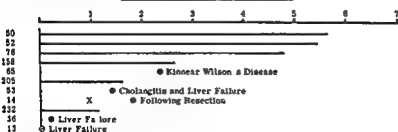
MODERATE CIRRHOSIS TIMES OF SURVIVAL AND FOLLOW UP IN YEARS FOLLOWING PORTA CAVAL ANASTOMOSIS

Total 33 Patients



AND FOLLOWING LIENO RENAL ANASTOMOSIS

Total 11 Patients



CAUSES OF DEATH AND FAILURE WRITTEN IN

OPERATIVE MORTALITY = 10 %
SUCCESS RATE = 90 %
Average Follow up = 2 years

KEY
— SURVIVOR
● LATE DEATH
○ POST OPERATIVE DEATH
X THROMBOSED

FIG 7

and of the same size as when constructed. Properly made shunts do not, therefore, contract (Fig 98). The girl with Kinnear Wilson's disease survived with improvement in liver function for two and a quarter years after lieno-renal anastomosis, finally dying of her neurological disease.

Liver failure was completely or partly responsible for four out of the five post-operative deaths. The fifth was unfortunate. She recovered well from porta-caval anastomosis only to die of a septicaemia due to resistant staphylococcus combined with a candida albicans infection both derived from the alimentary canal and starting at the end of a course of aureomycin treatment (Matthias and Rees).

Splenectomy.

This was done six times. One done for anaemia was successful. The other five had all had their spleens removed before being referred for treatment of persistent haemorrhage and were therefore failures. (What is not known is the number of people who have their spleens removed elsewhere and are not referred because they do not bleed.)

Hepatic and Splenic Arterial Ligation.

This was done on two occasions, both for ascites, both four and a half years ago. Both have been reasonably successful, though one went through a period of considerable ill health on account of splenic necrosis. The residual sinus had to be excised, and she has been better since (Case No. 75).

4 ADVANCED CIRRHOSIS (110 patients)

Only twenty-one of the patients in this group were alive at the time of the analysis, a fact which adds emphasis to the terrible nature of the disease. Sixty had had no operation and of these forty-seven are dead, mostly of liver failure. F
treatment . . .
anastomosis done since the analysis with one death

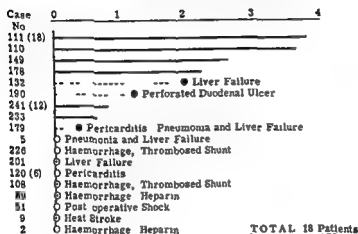
Porta-caval and Lieno renal Anastomosis.

Twenty three patients have had either porta-caval or lieno-renal anastomosis and the results are set out in the table (Fig 73). Compared with cirrhosis of less severity the results are depressing but there are a few remarkable successes which make surgical treatment worth while in certain carefully selected cases. Included are four early patients Nos. 2, 5, 9, and 11, operated on in 1948 and 1949 who would not at present have been considered suitable for operative treatment. The operative mortality thus includes all patients

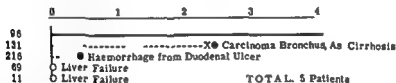
OPERATIVE TREATMENT OF PORTAL HYPERTENSION

operated upon during what may be termed the probationary period between 1947 and 1951. Since then much greater care has been taken in the pre operative treatment and selection

SEVERE CIRRHOSIS TIMES OF SURVIVAL AND FOLLOW-UP IN YEARS FOLLOWING PORTA-CAVAL ANASTOMOSIS



AND FOLLOWING LIENO-RENAL ANASTOMOSIS



CAUSES OF DEATH WRITTEN IN

OPERATIVE MORTALITY = 40%
SUCCESS RATE = 44%
Average follow-up = 2 years

KEY -

— = SURVIVOR
- - - = Late Death
O = Post-operative Death
X = Thrombosed

FIG 73

This includes many early failures e.g. Cases 2, 5, 9 and 11 who were operated on in 1948 and 1949 when the limitations of operative treatment were not recognised

Only seven of the twenty three survive in good or reasonable health but the success rate is given as 44 per cent because two died of intercurrent disease (Patient No 190 of perforated duodenal ulcer and Patient No 216 of haematemesis from duodenal ulcer) and one of subacute hepatitis two years

after making a remarkable recovery from an emergency porta-caval anastomosis for haemorrhage in the presence of ascites and coma (Patient No 132) At autopsy all these shunts were satisfactory On the other hand, an arsenical cirrhotic who survived two and a half years after lieno-renal anastomosis and died of a carcinoma of the bronchus, was found at autopsy to have a thrombosed shunt and is therefore considered a failure

Splenectomy.

This was done six times with four deaths One done for severe anaemia and ascites has been successful The other survivor was a failure on account of continued haemorrhage A porta-caval anastomosis has been done since the analysis and he is now well and at full work

Proximal Gastric Resection.

This has been done once The patient died seven weeks later

Hepatic Arterial Ligation.

This has been done on twelve occasions Eleven patients died post operatively and one nineteen months later of haemorrhage and liver failure (Case No 61) The comparison with shunt operations is not valid because arterial ligation was done for the more serious cases with ascites who were considered unsuitable for porta-caval anastomosis

Other Operations.

Various other operations have also been done, the button operation once (Crosby and Cooney), peritoneal saphenous anastomosis once, and the Talma Morison operation on four occasions, but none has been of any lasting value Intrapertoneal adhesions which form following the Talma Morison operation make all subsequent abdominal operations doubly difficult and occasionally impossible

Arterialisation of the Liver. (Portal Systemic Exchange.)

The depressing procession of deaths from liver failure, particularly those following many of the early porta-caval anastomoses confirmed the opinion that a patient with a severely damaged liver could not stand up to the effects of the cutting off of the portal supply of blood It seemed that if arterial blood could replace the portal loss the patients might survive On an occasion in 1949 a patient was dying in hepatic coma following lieno-renal anastomosis A second operation was done as expeditiously as possible in which the right kidney was removed, the portal vein divided and its hepatic end anastomosed to the right renal artery The patient's life was not saved An operation was

OPERATIVE TREATMENT OF PORTAL HYPERTENSION

then devised for the simultaneous decompression of the portal tree and arterialisation of the liver by constructing an end to side porta-caval anastomosis and then anastomosing the hepatic end of the portal vein to the right renal artery end-to-end (Hunt, 1952) This operation was done on three patients in the terminal stage of cirrhosis in 1950, but all died during the post operative phase, two of liver failure and one of retroperitoneal haemorrhage due to heparin administration The operation was therefore abandoned, but it may yet be possible to devise some such procedure for the advanced case Cohn and Herrod, and Russ Fisher, and their colleagues have entertained

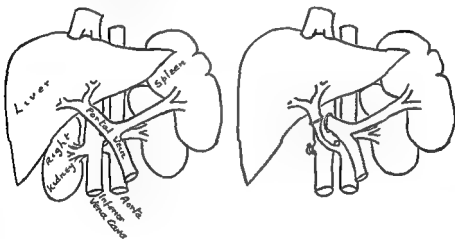


FIG 74

Arterialisation of the liver by the operation of portal systemic exchange porta-caval and reno-portal anastomoses

similar ideas and done valuable experimental work Leger, Albot, and Vaile have considered the opposite procedure of 'portalisation of the hepatic artery,' but this amounts to little more than the hepatic arterial ligation of Rienhoff

In summary the results of operative treatment in portal hypertension due to cirrhosis have been very satisfactory when porta-caval anastomosis has been necessary and possible in cases of mild or moderate cirrhosis Lieno-renal anastomosis has been less satisfactory Splenectomy has occasionally been effective on its own Proximal gastric resection and transection have succeeded only in cases of mild cirrhosis The major problem of advanced cirrhosis remains These patients account for more than half the cirrhotics and only a privileged few survive with treatment and return to work Of these the shunted patients seem to be the most satisfactory They can, of course, only be operated on if they improve under intensive medical therapy, so credit must go equally to medical and surgical treatment for their recovery and well-

being. Other operations are of little value, though there may be a place for hepatic arterial ligation. Further details of the precise choice of operation will be discussed after the complications and effects have been considered.

Lastly, in animal experiments on the regenerative power of the liver it has been shown that those with deprivation of portal blood are the least successful—50 per cent regeneration—and those with arterial replacement of portal blood the best—slightly more than 100 per cent regeneration. Ravdin reviewed the situation in his Moynihan lecture at the Royal College of Surgeons in December 1956. The findings add emphasis to the suggestion that further developments in the surgical treatment of advanced cirrhosis may well be along the lines of arterialisation of the liver.

CHAPTER XIII

THE COMPLICATIONS OF OPERATIVE TREATMENT

THE complications of operations for portal hypertension are of three main types (1) Those of any major upper abdominal or abdomino-thoracic operation (2) those concerned with the portal and splenic veins and (3) those specifically related to liver dysfunction remembering that any interference with the liver renders the rest of the body less vigorous. For example reduction in the serum proteins will delay the proliferation of fibroblasts and make the incisions more liable to *disruption* a complication which occurred three times in the whole series of 322 major operations. Secure suturing with inert non absorbable material is imperative as a precautionary measure. Also the oedematous intestine often associated with ascites will impede the absorption of nutriment and contribute to a state that can only be called concealed malnutrition. It is not possible therefore to distinguish precisely between the non specific and the specific complications.

1 Infective Complications

PULMONARY—Pulmonary collapse bronchitis and broncho-pneumonia are common and precautionary measures are therefore taken as a routine. There has also been one case of lung abscess and one of empyema which followed the reconstruction of an oesophago-gastric anastomosis.

WOUND INFECTIONS—The wounds for these operations are often large. They sometimes become angry and inflamed without developing suppuration. The temperature becomes raised and may persist so for three or four weeks. Cause for the fever is sought all over the body and may not be found. Since penicillin and streptomycin have been given prophylactically for the five days after operation this troublesome condition of the wound has been seen much less frequently.

Peritonitis and subphrenic abscess have not occurred though they have been diagnosed in error on three occasions.

Suppurative pericarditis accounted for the death of one patient during the post-operative phase but this was not considered to be a direct consequence of the treatment.

2 Abdominal Distension

This may give trouble and persist for more than two days post-operatively. If it increases and is associated with colicky spasms and inability to pass wind subacute intestinal obstruction due to mesenteric venous thrombosis must always be considered.

3 Mesenteric Thrombosis.

All degrees of mesenteric thrombosis exist. The full or textbook picture, with incipient gangrene of the intestine, may take five days to develop. It has been encountered five times, but only once during the post-operative phase—following an exploratory operation—and never after porta-caval anastomosis. Persistent abdominal distension, with a low fever and moderately elevated pulse rate, has proved later on three occasions to have been due to mesenteric thrombosis. The condition, as emphasised by Bussey, need not therefore be catastrophic. The usual treatment for incipient paralytic ileus has always been adopted, omitting only aspiration by a Ryles tube owing to the risks of haemorrhage from oesophageal erosions. Recently heparin in carefully controlled doses has been administered for this persistent distension with apparent benefit. Disturbed intestinal motility only becomes evident when the mesenteric vein itself is involved in the thrombotic process.

4 Thrombosis of the Anastomotic Stoma.

The clinical picture of thrombosis of a lienorenal anastomosis with extension of the clot throughout the splenic and portal veins without involvement of the mesenteric, was given by a recent patient whose venograms are shown in Fig 13 and whose chart is given on Fig 75. He appeared to be recovering well on the third day, but from then on he deteriorated and developed a persistent fever which became worst on the tenth post-operative day. He became very anaemic, at first without apparent external or internal blood loss, but with melaena later which was in no way proportional to the amount of blood that had to be transfused. Other physical signs and radiological findings were negative. Melaena and anaemia persisted, so the abdomen was re-explored. The only abnormality discovered was the thrombosed portal and splenic vein, which felt like a large hard rod. There was no other cause for his pyrexia. The portal vein was divided and the clot excavated to enable a porta-caval anastomosis to be constructed. His subsequent course has been excellent.

Obliteration of the shunt has been assumed to have occurred if the patient has further gastro-oesophageal haemorrhage. Very occasionally the bleeding has come from a duodenal ulcer in the presence of a good anastomosis. Splenic venography or other indirect methods of proving patency have never been used owing to the discomfort and risk involved, slight though it is. Patients who have recovered from shunt operations and had no further haemorrhage or ascites are considered to have patent anastomoses, and this has been the case with but two proved exceptions, a girl with congenital portal vein obliteration and a man with advanced arsenical cirrhosis. On all other occasions when the opportunity has arisen, clinical assessment of patency has been

COMPLICATIONS OF OPERATIVE TREATMENT

confirmed at incidental abdominal operation by portal venography or after death by postmortem examination

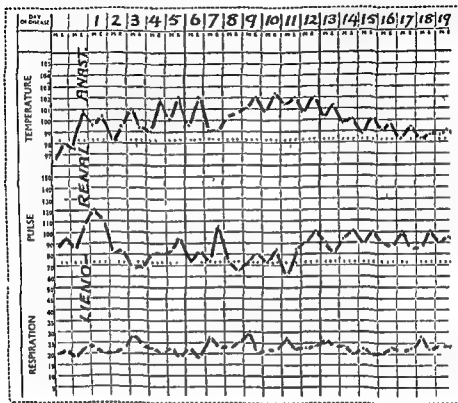


FIG 75

"Post-splenectomy Fever."

Case No 242 The chart of a case of portal and splenic vein thrombosis

The figures for thrombosis are as follows —

(1) In cases of congenital obliteration or stricture of the portal vein thrombosis occurred in

7 out of 10 lieno-renal anastomoses (70 per cent)

5 " " 5 'make-shift' " (100 per cent)

0 " " 3 porta-caval " (0)

The reasons for the high percentage have already been given (p 101)

(2) In cases of acquired thrombosis of the portal vein thrombosis occurred in

1 out of 4 lieno-renal anastomoses

- (3) In all cases of cirrhosis hepatic thrombosis occurred in
 4 out of 28 lieno renal anastomoses (14 per cent)
 4 " " 68 porta-caval " (6 per cent)

Blakemore, analysing 111 shunt operations in 1952, gives the figure for thrombosis as follows spleno renal, 11.5 per cent, porta-caval, 6.5 per cent and small veins, 55 per cent

Porta-caval anastomosis is without doubt the better method. Of the four cases that have thrombosed, one was a child of five with a small portal vein, one had a long thin portal vein (Fig. 38A), in the third the portal vein was kinked over an abnormal hepatic artery and the fourth already had a thrombosis of portal and splenic veins when the shunt was constructed. The corollary to these findings is that *no properly constructed porta-caval anastomosis has yet clotted up*. This result justifies the taking of minute care in the construction of stomata. (See Appendix 2)

Lieno renal anastomosis in adults is better than in children but not so satisfactory as porta caval. The vein is more delicate, longer, made to take a more tortuous course, more liable to be kinked, and has the direction of blood flow reversed. Lastly, splenectomy predisposes to thrombosis through the elevation of the platelet count (Graham Bryce). Removal of the spleen is a prerequisite of lieno renal anastomosis.

In comparing end to side with end-to-end anastomoses the proportion that have thrombosed is three in twenty-one and one in seven respectively, so one method has no advantages over the other in this respect.

Anticoagulant therapy is not now used except in cases of suspected mesenteric or intrahepatic thrombosis and when occlusion of an anastomotic stoma can be anticipated, e.g. after clot has been scraped from the portal vein. When it has to be given, it should be used with great circumspection and preferably not within twenty-four hours of operation. It was used more freely in the early cases and led to four deaths from retroperitoneal haemorrhage. No anastomotic stoma has leaked, even with heparin.

5 Episodic Stupor and Hepatic Coma (Fig. 76)

Episodic stupor following porta-caval anastomosis is the extreme of 'portal to systemic encephalopathy' (Sherlock, Summerskill, White, and Phear). McDermott and Adams' case in 1954 followed excision of a carcinoma of the pancreas with an involved segment of portal vein, but in the present series it has not been encountered in extrahepatic obstruction. The incidence has been as follows:

MILD CIRRHOSIS—21 patients who had venous shunts

- 1 late death in hepatic coma
- 2 cases causing incapacitating episodes of stupor
- 4 mild or transient episodes

COMPLICATIONS OF OPERATIVE TREATMENT

MODERATE CIRRHOSIS—47 patients who had venous shunts (5 post-operative deaths 3 of liver failure)

- 7 late deaths in hepatic coma
- 2 incapacitating
- 5 mild or transient episodes



FIG. 76

CASE No. 93. The vacant, puzzled, trusting and sad look of the patient episode of stupor. Confused and apathetic.

ADVANCED CIRRHOSIS—23 patients who had venous shunts (11 post-operative deaths)

- 1 late death in hepatic coma
- 3 mild or transient episodes

In cirrhosis as a whole therefore hepatic stupor appeared during or following convalescence from portal to systemic venous anastomosis in all degrees of severity and chronicity in twenty-five out of ninety-one patients operated on (27.5 per cent). In half the attacks are trivial. In contrast to these figures Blakemore states that he has seen intoxication in only two or

three instances in 234 shunted cases (in the discussion to Patton, Lombardo, and Lyons' article)

Of the nine late deaths (9.9 per cent of all), seven patients died of progressive liver disease, one of carcinoma of the liver, and one of cholangitis. The terminal coma in each patient began as episodic stupor. In most of these patients the porta-caval anastomosis was well worth while and certainly prolonged the usefulness of life. In some the temporary recovery brought great happiness. The operations were not failures from the patients' point of view.

Four patients are incapacitated (4.4 per cent). Three are female and in the older age group and the fourth is an anxious man worrying because he can no longer do the responsible work for which he is trained. The extent of associated psychological disturbance is sometimes difficult to assess and electroencephalography may assist in diagnosis.

Twelve patients (13.2 per cent) are in the mild or transient group. Three have had but one brief attack of stupor within a few weeks of porta-caval anastomosis. The others have had a varying number of attacks, most of which are related to the consumption of more protein than can be tolerated. One patient has a duodenal ulcer which has bled on two occasions, each inducing stupor, otherwise she is better with a high protein diet.

No common denominator has been discovered. Stupor is not confined to the more advanced cirrhotics, though it is more likely to develop in those with the poorer liver function. For example, of the mild cirrhotics only two patients had serum albumin levels of less than 4 gm per 100 ml pre-operatively, and both are now incapacitated. It occurs following both porta-caval and sieno renal anastomosis. Sometimes it appears that the greater the flow of blood to the liver pre-operatively, the greater the tendency to liver failure or episodic stupor after porta-caval anastomosis, and, conversely, the greater the natural diversion of portal blood (as shown by venography), the less the tendency to post-operative stupor. This is notable when the portal vein is thrombosed. As Blakemore (1956) suggests, if the liver has little or nothing to lose from a shunt, it is unlikely to suffer from diversion of its portal supply of blood.

Incapacitating attacks have continued for more than three years in two patients, each attack necessitating treatment in hospital, yet the patients have recovered reasonable health on each occasion and the liver functions are only slowly deteriorating. The treatment of the severe attack is the same as that of hepatic coma (see page 92).

The management of the patient with mild episodes is of great importance because he can keep at his work provided he takes an intelligent interest in his condition. At the same time everything should be done to prevent him becoming introspective. He must adhere to a suitable low protein main-

COMPLICATIONS OF OPERATIVE TREATMENT

ance diet (45 to 60 gm per day) and reduce the protein content even further as soon as he appreciates that an episode is beginning. Most are content to become almost vegetarians, with fruit, vegetables, and salads the basis of their diet. Many benefit from tetracycline and keep with them a supply of twenty 250 mg tablets for a five-day course together with vitamin B concentrate. Some maintain that sodium glutamate is of value and do not need to be persuaded to take it, others find amphetamine sulphate helpful in keeping them awake and at work, and others take paraldehyde or chloral hydrate for the night insomnia which so often accompanies drowsiness by day. Their work should carry no responsibility, be quiet, and not strenuous. They should carry on their persons (1) an identity note in case they develop stupor while travelling (which is the time when their well regulated existence is most likely to be disturbed), and (2) a medical note, including the telephone number of an accessible physician or surgeon familiar with their case. Mistaken charges have been levelled against patients in incipient stupor and faulty treatment given.

6 Diabetes.

On each of the three occasions that diabetes has been encountered post-operatively it has caused great difficulties. The symptoms of diabetic coma have faded into those of hepatic stupor and stabilisation has been difficult. That it occurs as a complication of portal hypertension must be recognised because of the risks of giving inappropriate treatment, *e.g.*, glucose in the mistaken belief that the coma is hepatic in origin.

A possible clue to its cause was discovered in a fourth case of hyperglycaemia which developed in a patient too ill for operation. She died and at autopsy the portal, splenic, and pancreatic veins were diffusely thrombosed suggesting that the resultant congestion was a causative factor. Two of the three post-operative diabetics had thrombosis of the portal vein at operation (Fig 48 p 55). The third died later and haemochromatosis was conclusively demonstrated at necropsy.

7 'Post-splenectomy Fever.' (Figs 13 and 75)

This is a condition for which a cause must exist. Every attempt should be made to find it by purposeful examination of lungs and pleura, abdomen and subphrenic spaces, urine and perinephric spaces and the wound itself. The liver and the biliary passages should be considered when jaundice is present. Lastly portal and splenic vein thrombosis must be kept constantly in mind but should not be diagnosed too readily or before infective conditions have been excluded.

8 Icterus.

This sometimes appears or increases following porta-caval anastomosis. Occasionally it heralds the beginning of progressive liver failure, but more often it is an independent phenomenon which interferes but little with the patient's work or life.

9 Oedema of the Ankles.

Oedema of the ankles, of a uniform and persistent type, has been encountered five times in patients with successful porta-caval anastomosis having been absent beforehand. It can be alleviated by the use of elastic bandages and stockings. The cause, in the absence of a reduction in the level of serum albumin, may well be the increase in hydrostatic pressure within the inferior vena cava.

10 Obstructive Jaundice.

Jaundice due to inadvertant occlusion of the common bile duct at operation has occurred three times, once in attempting a 'makeshift' porta-caval anastomosis in a girl with extrahepatic obstruction after the spleen had been removed elsewhere (Patient No. 49), once in exploring a thrombosed portal vein in a patient with cirrhosis whose spleen had been removed thirty years before (Patient No. 62), and once during the course of a difficult porta-caval anastomosis for advanced cirrhosis. The jaundice has been relieved in the first case by choledochojejunostomy and in the second by cholecystojejunostomy. The third patient died on the eleventh post-operative day of liver failure. Only good venograms to show the exact state and position of the portal vein and practice can minimise the risks of these accidents but it is not possible to be certain of avoiding them in some exceptionally difficult operations.

CHAPTER XIV

THE EFFECTS OF PORTAL TO SYSTEMIC VENOUS ANASTOMOSES

THE frequency of the various complications and their occasional devastating effects must not be allowed to conceal the fact that the results of venous anastomoses have been excellent in 86 per cent of the mild cirrhotics operated on, 70 per cent of the moderate, and 44 per cent of the severe, figures that are comparable with those given by Senn and Blakemore. These patients have recovered good health and been able to return to normal or almost normal lives, free from the threat of haemorrhage and relieved of ascites. Many have gained in weight and strength; some have returned to strenuous occupations—farming, riding, playing football, working in out of the way parts of the world and so on. Even some of the 'failures' have returned temporarily to their usual work. One such, Patient No. 78, from being bedridden with haemorrhage and ascites, was able to go back to being captain of a merchant ship for two years, finally dying of presumed liver failure more than three years after his operation.

COLLAPSE OF OESOPHAGAL VARICES

The most urgent necessity of a shunt operation is to stop haemorrhage, and this it does if the anastomosis is of adequate size and well constructed, by causing the oesophageal varices to collapse (Figs 77 and 80). After operation three weeks or more should be allowed to elapse before attempting to demonstrate this by barium swallow because the change is not instantaneous. When the veins are old and rigid they cannot ever disappear, but the risk of serious haemorrhage will be eliminated.

DECOMPRESSION OF PORTAL TREE

The shunt will drain blood away from the varices and into the inferior vena cava as shown in Fig. 78 (Patient No. 24). Both reproductions are of portal venograms: the second obtained at a subsequent operation for appendicectomy.

The patency of the shunts has not been tested by doing splenic venograms, but the magnificent cine radiographs shown by Sandblom and Milnes Walker would appear to justify the occasional use of this method to illustrate the whole process of the filling and emptying of the portal tree. The only tests of patency that matter are the prevention of haemorrhage or relief of ascites.



FIG 77

Barium swallows to show how oesophageal varices shrink after porta caval anastomosis: A before operation, B after (A B Case No 259 A II Case No 146)

EFFECTS OF PORTAL TO SYSTEMIC VENOUS ANASTOMOSES

Similarly, rapid evacuation of the portal system can be demonstrated conclusively by the use of radioactive sodium and the double scintillation detector. Fig 79 shows two pairs of graphs, before and after porta-caval and lieno renal anastomosis. Two differences can be detected in each pair, especially the second (1) the increase of speed, and (2) the reduction in the time taken for the radioactive material to be washed out of the portal or splenic vein. The graphs thus also indicate the extent to which stagnation of blood has been relieved.

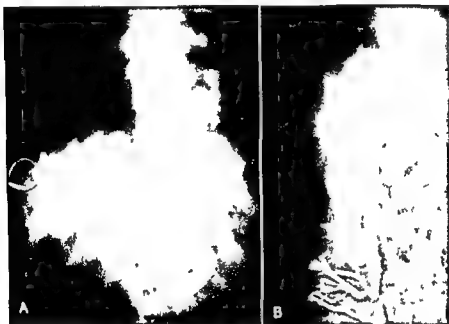


FIG 78
Portal Venograms

CASE No 24. A. Before porta-caval anastomosis to show congestion and retrograde flow in oesophageal varices. B. After porta-caval anastomosis to show how completely the portal tree is decompressed.

ELIMINATION OF ASCITES

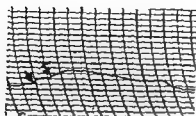
It is difficult to understand the current tendency to view with disfavour any attempt to treat ascites surgically when confronted with evidence such as the photographs of the abdomen in Figs 80a (Case No 205) and 80b (Case No 250). If medical treatment fails and the liver function is sufficiently good a shunt is the correct treatment whatever theory on the aetiology of ascites is most in favour at the time. The patient illustrated in Fig 80

PORTAL HYPERTENSION

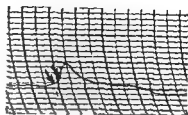
had been bedridden for months while under medical treatment, yet returned to domestic work three months after lieno renal anastomosis and has continued at work since, with time off for a Wertheim's hysterectomy for carcinoma of the cervix. Altogether twenty patients with gross ascites and two with ascites

Case No 142

S 34 ♂, 27.11.55 Portal vein



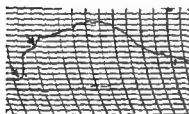
Before anastomosis 4.3 cm/sec



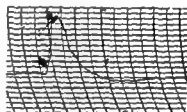
After anastomosis 12.5 cm/sec

Case No 201

P 49 ♂ 8.2.55 Portal vein



Before anastomosis 2.5 cm/sec



After anastomosis 9.0 cm/sec

B
FIG 79

Two graphs to show the speed of flow in the portal vein before and after porta caval anastomosis. Stagnation of blood containing radioactive sodium is present before increased speed and more rapid clearing of the portal vein after.

which had improved with medical treatment have recovered from porta-caval or lieno renal anastomosis and been cured of their ascites. Thirteen had haemorrhage in addition. Five have died: two of carcinoma of the bronchus, one of perforated duodenal ulcer, and two of liver failure, and none developed ascites as a terminal event though this can occur. Even if the obstruction in ascites is in the outflow from the liver, as suggested by Madden *et al* in 1954, there is no reason why relief of the hypertension should not produce an improvement. Blakemore (1949) describes successful relief of ascites in a case of Budd-Chiari disease.

EFFECTS OF PORTAL TO SYSTEMIC VENOUS ANASTOMOSES



FIG 80a

Case No. 205. The abdomen before and after lipo-renal anastomosis for intractable ascites. The scars of repeated paracenteses can be seen. Syphilitic cirrhosis. (See Fig. 39.)

THE EFFECTS OF OPERATION ON BLOOD COUNTS AND LIVER FUNCTION

Blood counts and significant liver function tests have been done at regular intervals since operation and related to the course of each patient. Usually they have confirmed the clinical impressions. To attempt to assess in precise mathematical terms the state of the blood and the liver has however, been difficult because the fluctuations in the readings obtained on consecutive visits have often been considerably greater than the difference before and after operation. Macpherson, Owen, and Innes, in very thorough statistical

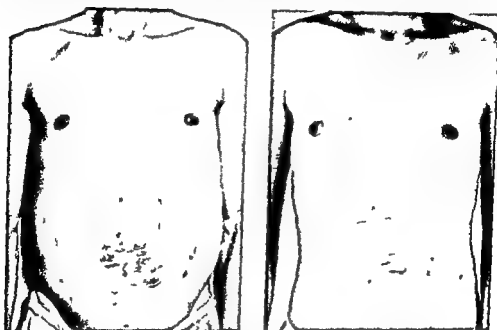


FIG. 30a

Case No. 280. The abdomen before and two months after porta caval anastomosis for haemorrhage and intractable ascites. Ascites has gone abdominal veins collapsed and nutrition improved.

analyses, allow for this error. They point out that deviation of blood from the cirrhotic liver did not cause any significant deterioration of liver function in six patients three years after operation, and they concluded that the survival rates of all cases treated surgically are significantly better than after medical treatment. Ellis, Linton and Jones show that serum albumin improves in 29 per cent, remains the same in 53 per cent and becomes worse in 18 per cent.

In the present series no searching statistical analysis has yet been attempted but the overall figures may be of some interest. The figures for liver function tests are the averages of all cases, successes and failures. The haemoglobin levels are necessarily taken when there has been no recent haemorrhage.

EFFECTS OF PORTAL TO SYSTEMIC VENOUS ANASTOMOSES

EXTRAHEPATIC OBSTRUCTION

HAEMOGLOBIN	8 patients	Before	porta-caval or lienorenal anastomosis or splenectomy	78 per cent
		After	" "	104 per cent
WHITE BLOOD COUNT	12 patients	Before	" "	3,600
		After	" "	8,700
PLATELETS	12 patients	Before	" "	77,000
		After	" "	439,000

MILD CIRRHOSIS

HAEMOGLOBIN	7 patients	Before	venous anastomosis	72 per cent
		After	" "	102 per cent
SERUM ALBUMIN	20 patients	Before	" "	4.6 gm / 100 ml
		After	" "	4.5 gm / 100 ml
PSEUDO-CHOLINESTERASE	16 patients	Before	" "	63 units
		After	" "	75 units

In this group the white blood count and platelets have shown considerable increase following porta-caval anastomosis, 2,000 to 8,000 and 160,000 to 400,000 being expected figures

MODERATE CIRRHOSIS

SERUM ALBUMIN	26 patients	Before	venous anastomosis	4.2 gm / 100 ml
		After	" "	4.0 gm / 100 ml
PSEUDO-CHOLINESTERASE	26 patients	Before	" "	54 units
		After	" "	58 units

ADVANCED CIRRHOSIS

SERUM ALBUMIN	16 patients	Before	porta-caval anastomosis	3.6 gm / 100 ml
		After	" "	3.5 gm / 100 ml
PSEUDO-CHOLINESTERASE	14 patients	Before	" "	45 units
		After	" "	46 units

Of the many not fit for operative treatment, the serum albumin was 3.0 gm / 100 ml (average of fifty five patients) and the pseudocholinesterase 30 (average of forty seven patients), figures below the critical level

When deterioration has led to death from liver failure, the decline in function tests has usually been evident. Patient No. 166, a moderate cirrhotic, had a serum albumin level of 4.8 gm and pseudocholinesterase of 63 units pre-operatively. They fell to 3.1 and 54 post-operatively, rising at four months to 4.1 and 63, then falling away over the months—3.7 and 35, 3.3 and 31,

26 and 35—prior to terminal deterioration and death at two years and one month after operation. During this whole period the serum globulin never fell below 3 or rose above 3.7.

The serum albumin level in Patient No. 78 was 3.9 before porta-caval anastomosis. 2.5 after, rose to 3.5, and then slowly declined to 1.8 prior to his terminal illness.

Improvement, when it has been demonstrable, has been most marked in the severe cirrhotics. Patient No. 233 had a serum albumin level of 2.2 gm and pseudocholinesterase of 34 units at one time during his pre-operative course of medical treatment. When well, six months after operation the figures were 4.2 and 66 respectively. The progress of Patient No. 149 is set out on the graph (Fig. 68, p. 94). The improvement under intensive medical treatment was impressive in spite of repeated haemorrhages and except for the expected drop in function immediately after porta-caval anastomosis, it has been well maintained since.

Following ilio-renal anastomosis also there is often demonstrable improvement of liver function, e.g. in Patient No. 205 the serum albumin rose from 3.7 to 5.2 gm and pseudocholinesterase from 51 to 85 units (this patient was a proved syphilitic).

Two patients surviving hepatic arterial ligation have serum albumin levels of 5.2 gm four and a half years after operation. The figures rose from 3.4 and 3.5 respectively before operation. Another patient who survived for nineteen months after hepatic arterial ligation also had a remarkable improvement in liver function from a serum albumin level of 3.2 to 4.25 gm. Her progress is set out on the graph (Fig. 69, p. 95).

The serum bilirubin has rarely become less post-operatively, unless an obstruction to the common duct has been removed. In two patients with advanced primary biliary cirrhosis it has become slightly more elevated while there has been little change in the other liver function tests.

Reduction of liver function was always noticed in the immediate post-operative period whether the operation was for portal hypertension or not, e.g., a fall in serum albumin from 5 to 1.5 gm/100 ml following Polya gastrectomy for duodenal ulcer in the presence of mild cirrhosis.

In assessing the progress and prognosis of individual patients the assistance given by the different tests has been similar to their help in pre-operative assessment—the serum albumin providing the solid reliable test and the pseudocholinesterase reflecting minor changes in day to day values and giving earlier warning of serious developments, e.g. the appearance of hepatitis in one patient was accompanied by a reduction of serum albumin by only 0.2 gm, from 4.1 gm/100 ml to 3.9, but of pseudocholinesterase by 50 units from 95 to 45.

EFFECTS OF PORTAL TO SYSTEMIC VENOUS ANASTOMOSES

The tests, therefore, confirm the findings of others that some patients improve and some deteriorate following shunt operations. The surgeon hopes that he will one day be able to distinguish pre-operatively between these two groups. It is easy to understand that cessation of an inflammatory or degenerative process within the liver will result in an improvement of liver function so long as other factors such as haemorrhage do not intrude. What is difficult to comprehend is how positive improvement can come about following a porta-caval anastomosis, especially in view of the animal experimental evidence which shows that portal blood is essential for the regeneration of liver tissue (Gray, 1951). Other workers, however, have cast doubt on these conclusions (Weinbren), and Mann, Wakim and Baggenstoss in 1953 have shown that the regenerating nodules are supplied by hepatic arterial blood only. This confusion of evidence from the laboratories must not allow the fact to be overlooked that in the human patient the liver can sometimes improve in function following deprivation of its portal blood supply. Nor should the even more surprising finding be forgotten that a similar improvement can occasionally occur after hepatic arterial ligation.

The overall conclusion is self-evident, that our choice of operation is at present haphazard and that there is no strictly scientific guide as to what we should do and when we should do it. Looking to the future it seems probable that further study of the microscopy of the liver cells themselves and their intimate relations with the finer branches of the hepatic artery and portal vein may help in making our selection of operation more logical. The beautiful three-dimensional studies of Elias have not yet been translated from the anatomical into practical pathological terms. It seems inevitable that fresh discoveries will be made when pathologists have forgotten about liver columns and accept that the liver cells are arranged in a continuous series of intercommunicating laminations—the membranous syndesmosis of Elias—which become broken, deformed and thickened in disease. For the present, however, we are compelled to act on the evidence that is to hand: clinical appraisal and overall tests of liver function.

CHAPTER XV

THE CHOICE OF OPERATION IN THE LIGHT OF THE RESULTS OBTAINED

IT is now generally accepted that porta-caval anastomosis is more efficient than lieno renal at decompressing the portal tree and that it is less likely to thrombose. It is usually, therefore, the operation of choice, but there are times when lieno renal is the better, apart from those cases of extrahepatic obstruction where there is no portal vein. For example, a young person with mild cirrhosis may have a congested spleen of such a great size that it is an impediment to an efficient porta-caval anastomosis. It may also be greatly overactive and causing a reduction of platelets to levels which are dangerously low and may contribute to excessive haemorrhage at operation. It is well known that splenectomy in such circumstances will allow the platelets to return to the circulation quickly, sometimes within minutes (Lahey and Cyr). A pre-operative platelet count of 50,000 or less should, therefore, suggest splenectomy and lieno renal anastomosis if the spleen extends to below the level of the umbilicus or is otherwise proportionately enlarged.

The anaemia of hypersplenism also may preclude the efficient building up of a patient for a major shunt operation, and it is sometimes necessary to remove the spleen in order to render the patient fit for the more difficult porta-caval anastomosis to follow.

The conditions mentioned so far have been the comparatively exceptional reasons for advocating the less efficient operation when both portal and splenic veins are available. The choice is deliberate. It should be noted that during the earlier years the inclination was to do a lieno renal anastomosis more frequently than now, because it is an operation from which there is a retreat. Porta-caval anastomosis on the other hand can lead one into difficulties where there is risk of damage to surrounding vital structures and where there may be an immediate threat to life. Greater troubles arise when the vein chosen is found to be unsuitable or difficult. For example, a portal vein may be partly occluded by clot plastered against its wall and yet show up well on splenic venography. This state of affairs may only be discovered on beginning to free the portal vein from its bed. The surgeon will then have to decide if it is wise to persist with a dissection which may develop into one of exceptional difficulty and danger or if he should switch to lieno renal anastomosis. From experience the latter is the wiser course. If the thrombosis is discovered early in the operation and if the patient is in a position on the operating table so that either operation is feasible, the alternative lieno renal anastomosis should be done immediately. If much operating time has already

THE CHOICE OF OPERATION

been spent it is wise to close the wound and defer the lieno-renal shunt for two or three weeks. The reverse of this situation—the construction of a porta caval rather than a lieno renal anastomosis—does not pose so difficult a problem.

Retreat from the construction of a porta-caval anastomosis because of thrombosis does not infer that a thrombosed vein cannot be cleared of clot and used for a shunt. This has in fact been done on ten occasions, nine



Fig 81

Case No. 8. Porta caval anastomosis in continuity (Mangot, R. (1955). *Abdominal Operation*. 3rd ed. New York: Appleton Century Crofts Inc.)

porta-caval and one lieno-renal. Only one porta-caval and the lieno renal have thrombosed. Six porta-caval have been completely successful. In five the spleen had previously been removed and the portal vein was the only possible channel for a shunt and in the sixth the spleen was present but the dissection had progressed so far without undue difficulty that the anastomosis was completed. In the two remaining cases the shunt has remained fully patent but in one the patient died twenty one months after operation of

diabetic coma and in the other of liver failure on the third day after an operation of exceptional difficulty. There is no doubt that this last patient would have stood a better chance if the change had been made to hepato renal anastomosis early in the operation.



FIG 82

Case No 5. Porta-caval anastomosis in continuity. Hepatic part of portal vein thrombosed.

Apart from preliminary operations there have been seven occasions on which definitive treatment has had to be abandoned on account of exceptional technical difficulties. In addition there have been numerous diagnostic explorations mostly done elsewhere before the patients had been referred for definitive treatment.

THE CHOICE OF OPERATION

Mention has been made of the two types of porta-caval anastomosis, the end to-side and the side to side or anastomosis in-continuity (Fig 81) (de Martel, Rosenstein, Large *et al*) Many theoretical considerations based on the intention that some portal blood should be allowed to continue to flow to the liver, appear to favour the second type of anastomosis which has been

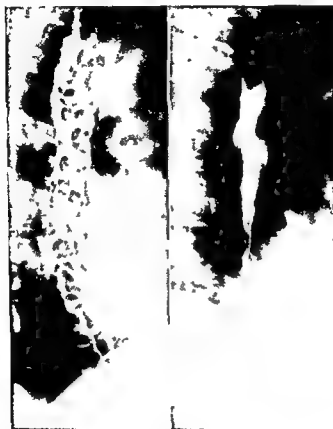


FIG 83

Patient No. 165: Barium swallow before and after successful proximal gastric transection

done three times in the present series. One patient was successful (No 7), though he died of carcinoma of the bronchus seven years later. At post mortem the shunt was found to be in perfect condition and both ends of the portal vein completely free from clot. Another patient died on the twenty seventh post operative day of Pfeiffer pneumonia (Case No 5). The hepatic end of the portal vein was found to be thrombosed (Fig 82), the shunt

PORTAL HYPERTENSION

functioning as an end to side anastomosis. The third patient (Case No. 8), who was considered a very good risk, died of liver failure unexpectedly and disappointingly in the post operative period. At the time of the operation it was noticed that a considerable volume of blood was flowing back from the liver towards the shunt and under considerable pressure. This blood must have



FIG. 84

Patient No. 183. Proximal gastric resection for traumatic thrombosis of portal vein. No varices but marked stricture.

come from the hepatic artery, so that the liver cells were not only deprived of their portal venous supply but also of some of their arterial blood, producing the reverse of the theoretical advantages of the method.

Mention must be made of hepatic arterial ligation (Rienhoff, 1951, Ber man and Hull, 1952, McFadzean and Cook, 1953), which is an operation that has been done on fourteen occasions in this present series. Two patients with moderate cirrhosis are alive and considerably improved. In both the operation was done for ascites. Oesophageal varices were not present. The remainder are all dead of liver failure or haemorrhage. Haemorrhage gener

THE CHOICE OF OPERATION

ally is accepted as an emphatic contraindication for the operation, and since it is the cardinal reason for surgical treatment of portal hypertension, there can be few occasions when the hepatic artery should be tied in preference to porta-caval anastomosis. Intractable ascites is the only indication, but reasonable liver function is a prerequisite of success and there is no guarantee



FIG 85

Case No. 266. Abdomen of patient with biliary cirrhosis after three abdominal explorations for jaundice. To show herniae, healed sinuses and vascularity from adhesions.

that haemorrhage will not subsequently occur (*vide* Case No. 61). It is probable, therefore, that the operation should be abandoned.

For patients with no portal or splenic vein some form of interruption operation should be done to stop haemorrhage. Injection of oesophageal varices (Crafoord and Frenckner, Macbeth) is a safe and comparatively simple procedure but cannot benefit gastric haemorrhage. Gastric transection has only been effective in one patient (Fig. 83, Patient No. 165). Proximal gastric resection has been effective in stopping haemorrhage in more than half

the cases in which it was used but it cannot be considered very satisfactory (Fig 84) On four occasions it has benefited children They do not gain weight as well as they should but they are not stunted Gastric substitution operations may prove more satisfactory than the simple resection done in this series (Appendix 4 A)

The Talma Morison omentopexy should never be done unless nothing else is possible It leaves the abdomen matted with excessively vascular adhesions which render any subsequent operation extremely difficult or quite impossible (Fig 85)

To summarise For all practical purposes the choice of operation rests between porta-caval or lieno renal anastomosis or some form of interruption However thorough the pre-operative investigation may be it is sometimes impossible to tell beforehand which will be the best for a particular patient The final choice may only be made after the operation has begun This leads directly to the study of the technical aspects of the surgical treatment of portal hypertension which are set out in Appendices 1 to 4

TECHNICAL CONSIDERATIONS

APPENDIX 1

THE PRELIMINARIES OF OPERATIVE TREATMENT

A The Position of the Patient on the Operating Table.

Flexibility of approach is essential. The hornpipe position (Fig 86) (Hunt, Lawrance, and Whiteley, 1956) has therefore been devised and is

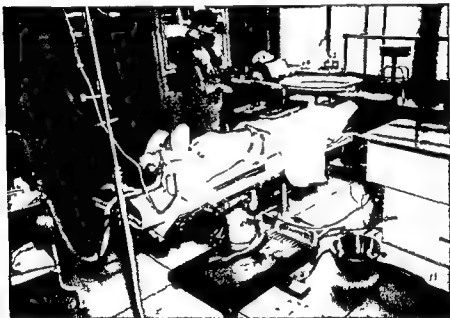


FIG 86

The hornpipe position. Note position of arms raised costal margin forward rotation of scapula freedom of approach from front and sides blood pressure cuff on right arm intra-venous needle in dorsum of left hand valves of endotracheal tube and double cassette tunnel ready for venography Arms are fixed with elastic adhesive strapping and in this figure left hand is supported on small pillow

always used. The patient is placed on his back on the operating table with the double cassette tunnel in place under him so that the film covers the whole area between the nipple and the umbilical planes. The arms are folded across the upper chest so that the hands come to lie more or less on opposite shoulders. They are held in place with adhesive strapping. Small pads may

be necessary for comfort and security. The advantages of this position are many. X rays can be obtained without interference. The surgeon is ideally placed to extend an abdominal incision out into either flank or up into the chest. There is nothing to obstruct the assistants' approach to the trunk. The subcostal angle is fully expanded, respiratory movements are not impeded and there is no risk of compression of any nerve of the brachial plexus. The anaesthetist, seated at the head of the table, is in full control of the patient's endotracheal tube, transfusion drip, and blood pressure cuff without having to move. With the minimum of trouble the patient can be rolled slightly on to his right side for a full transthoracic approach to the lower end of the oesophagus and cardiac end of stomach.

B Splenic Venography.

Splenic venograms if they are possible, are obtained as soon as the patient is settled in position on the operating table. The injection of 25 to 30 cc of 70 per cent diodone directly into the spleen is done as rapidly as possible, using a 50 cc syringe and an ordinary fine lumbar puncture needle. The point of insertion of the needle varies with the size and position of the spleen. Sometimes it is over the abdomen and sometimes through a lower inter costal space. The anaesthetist stops the breathing while the needle is pushed into the substance of the spleen, blood is aspirated to ensure that it is within the spleen, and the diodone forcibly injected. X rays are taken at the precise end of the injection and two seconds later. (Excellent technical details of this procedure are given by Walker, Middlemiss, and Nanson, and Atkinson Barnett, Sherlock, and Steiner.) The negatives are developed while the skin is being sterilised and the towels placed in position for operation. If the pictures obtained are satisfactory and provide clear information concerning the nature of the portal tree, the appropriate incision can be made with confidence beginning with the abdominal part.

If splenic venography cannot be done because there is no spleen or because the spleen is small or awkwardly situated, the operation and therefore the abdominal part of the incision is chosen on clinical grounds.

C Taking the Portal Pressure.

Fig 87 illustrates the method of taking the portal venous pressure and the necessary apparatus which consists of a cerebrospinal fluid manometer to which is attached a 300 mm length of rubber pressure tubing carrying a No 2 serum needle. The whole is filled with 3.8 per cent sodium citrate solution avoiding air locks and the tube is clamped. The pressure can be taken from any convenient radicle of the portal vein usually a jejunal. A vein is selected and the needle inserted over the intestine itself about 1 cm

APPENDIX 1

from the mesenteric attachment and run up into the vein as it lies in the mesentery. The clamp is then removed from the tube and the citrate



FIG 87

Method of taking the portal venous pressure. The assistant balancing off the meniscus so that the operator can read the pressure. Note needle inserted over wall of jejunum (Exploratory incision only)

allowed to run down the manometer. This is lowered and balanced off quickly against the portal venous pressure as shown in the figure. In order to ensure that there is no interference in the free communication between

the main portal venous trunk the radicle from which the pressure is being obtained and the citrate in the manometer, the anaesthetist compresses the anaesthetic bag for two or three seconds. If all communications are free the meniscus in the manometer will rise 5 to 10 mm giving proof of the accuracy of the reading. As the needle is removed a small mosquito clamp is applied to the intestinal wall at the point of the puncture and tied off with catgut.



FIG 88

Method of injecting diodone for portal venography. Note needle entering jejunal vein over wall of intestine. (Full length of incision with extension out into flank.)

D Portal Venography (With Dr G H du Boulay and Dr B Green)

Portal venograms are obtained if splenic venography has been impossible or if the splenic venograms are not considered completely satisfactory. The method adopted has been the simplest, quickest and easiest possible. The apparatus consists of an ordinary 50 cc syringe carrying finger and thumb grips to which is attached a No. 1 serum needle. It should be held as illustrated. A suitable jejunal vein is selected and the loop of intestine held in the left hand as shown in Fig 88. The needle is inserted over the intestine 1 cm from its mesenteric attachment and run up into the jejunal vein, the barrel of the syringe resting on the palm of the left hand to give it steadiness. 30 cc of diodone are then rapidly injected without altering the grip and the

X rays are taken at the finish of the injection and two seconds later. While the exposures are being made the hands of the operator are protected by a lead shield held over them by the assistant. The needle puncture is clamped and tied off as when taking the portal pressure.

Technically the method is smooth, steady, easy, and quick, and very rarely is one troubled by the beginnings of a mesenteric haematoma. Hazards are virtually eliminated. It is unnecessary to dissect up a large vein in order to run up a polythene tube into the portal vein for the injection of the diodone, as advocated by Child *et al* (1951).

E The Measurement of Speed of Flow. (With Prof W V Mayneord and Dr E H Belcher)

The method of determining the speed of flow in the portal or splenic vein is by the injection of a small quantity (0.1 cc) of isotonic sodium chloride solution containing 50 microcuries of radioactive sodium and observing the speed with which the head of this injection traverses a given length of portal or splenic vein. Its passage along the vein and the time taken for the radioactive sodium to be dispersed is recorded electrically (Figs 56, 57 and 79) by means of a double probe pointed scintillation detector electronically coupled to a pen recorder. The radiation dose to the patient is 0.1 r unit which is about a fiftieth of the dose of irradiation from a standard X ray exposure. The double scintillation detector is illustrated in Fig 89. The points are 7.5 cm apart. Fig 90 shows it sterilised, gloved, towelled and attached to the pen recording apparatus. In use for portal speed (Fig 92A) one of the points is placed



FIG 89

The Double Scintillation Detector used for the estimation of the speed of flow in the portal vein. (IVth Congrès de Gastro-Entérologie (1954). *L'Hypertension portale. Le Dumping Syndrome*. Paris: Masson et Cie.)

alongside the portal vein in the porta hepatis and the other at the junction of superior mesenteric and splenic veins. It does not matter whether the detector is pointing at or is alongside the vein. The venogram is obtained first so that the instrument can be accurately positioned. For the splenic vein the two points are placed in relation to the upper border of the pancreas (Fig 92B).

The method of injection (Fig 91) is very similar to that used in taking portal pressures and obtaining portal venograms

F Anaesthesia. (Most anaesthetics have been given by Dr R A Bowen)

The anaesthetic must be as little toxic to the liver as possible. Blakemore advocates cyclopropane, but the advantages of the diathermy are such that my preference is for something less explosive. Induction is usually carried out



FIG 90

The Scintillation Detector gloved and towelled for sterility. Pen recorder to the right

with a small dose of pentothal which is followed by gas, oxygen, and curare supplemented by further small doses of pentothal. An endotracheal tube is always inserted in order that full control may be maintained over the airway and that the patient may be fully oxygenated throughout the operation. The anaesthetist also controls the transfusion of blood which is always given, and an attempt is made to run in, during the course of the operation, as much blood as is lost in the operative field. This may be impossible when there is momentary profuse blood loss.

It has been shown by Shackman, Graber, and Melrose that general anaesthesia leads to a reduction of blood flow to the liver. Prolonged anaes-

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thetics are therefore to be avoided, and everything possible should be done to make the operation expeditious. It has been suggested from time to time that hypotension or hypothermia should be used to reduce haemorrhage in



FIG 91

To show injection of 0.1 cc radioactive sodium solution into a jejunal vein the point of the needle entering the wall of the jejunum about 1 cm from the mesenteric border. The hilt of the gloved and towelled Scintillation Detector is alongside the operator's left hand.

these difficult and dangerous operations but both methods have been shown to be accompanied by marked liver damage (Anlyan *et al*, Knocker, and Fisher *et al*) and have not, therefore, been tried out.

PORTAL HYPERTENSION

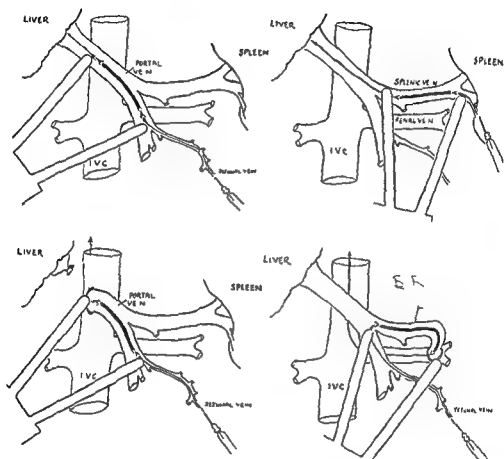


FIG 92

Method of measuring the speed of flow in the portal and splenic veins before and after portal systemic anastomoses using radioactive sodium and the Double Scintillation Detector (IV^e Congrès de Gastro Entérologie (1954) *L'Hypertension portale Le Dumping Syndrome* Paris Masson et Cie)

APPENDIX 2

PORTA-CAVAL ANASTOMOSIS

Credit for many of the technical advances in the surgery of portal hypertension by venous anastomoses must go to Blakemore of New York, Blalock of Baltimore, and Linton of Boston, Mass., but there are many surgeons on both sides of the Atlantic to whose work reference should be made for technical details. C. Stuart Welch in America, Santy and Marion in France, Learmonth of Edinburgh, and Milnes Walker of Bristol to mention but a few, and a recent well illustrated article by Hallenbeck of America. The operative methods that follow are derived in great part from some of the above sources. The application of these techniques to the circumstances of a surgical unit within a hospital is also important, and it has constantly been my aim to use methods which require little in the way of special equipment—with the one exception of the double scintillation detector, which serves a particular and limited research purpose. Otherwise all the technical equipment used is immediately available in any well stocked general operating unit, including the apparatus for portal and splenic venography and venous pressure readings. Long instruments and many retractors of all types, especially malleable retractors are of value. For the anastomosis itself and the preparation of the veins a few special instruments are of great help (Fig. 99), with appropriate fine, long, toothless dissecting forceps for handling the diminutive needles and delicate tissues in the bottom of a deep wound.

The initial exploratory incision is made from just above the umbilicus to the tip of the eleventh costal cartilage on the right side (Fig. 93). The abdomen is opened for the length of this incision, using diathermy for the deeper layers so as to avoid blood loss. The abdominal wall of a patient with portal hypertension may be excessively vascular, and it is essential to secure haemostasis as each layer is divided. The liver and spleen are examined and the gastro-hepatic omentum carefully palpated to define the position of the portal vein and hepatic artery if possible. The preliminary investigations as outlined in Appendix 1 are then completed and the feasibility of porta-caval anastomosis confirmed.

The incision is then extended outwards along the line of the eleventh rib on the right side. The rib and costal cartilage are resected and the diaphragm and peritoneum carefully incised. Sometimes the lower edge of the pleura in the costophrenic angle is never seen. On other occasions, if the dissection is done carefully, this edge can be found and pushed aside so that it is not damaged. In about one case in six the pleura is opened accidentally, and it should immediately be closed with catgut. Operating with the patient in the

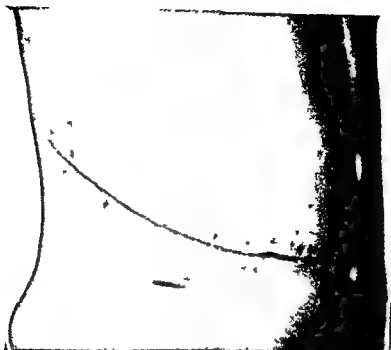


FIG 93

Case No 241 Incision for porta caval anastomosis from just above umbilicus across epigastrium and up along eleventh right rib



FIG 94

Case No 160 Transverse incision for porta caval anastomosis

dorsal hornpipe position resection of the eleventh rib allows the portal vein to be approached from the side, the surgeon sitting to his task. Blood does not collect in the wound because the incision extends back to level with the deepest point of the right kidney pouch. It is a comfortable incision from the point of view of surgeon and assistants (Fig. 88).

Satinsky advocates the full thoraco-abdominal approach with resection of the ninth rib, wide opening of the pleura, division of the diaphragm, and coronary ligament of the liver and dislocation of the liver into the chest. Milnes Walker uses a similar approach. Dislocation of the liver into the chest makes for easier access to the portal vein, but temporarily disturbs the normal anatomical relations.

Towels are affixed to the edges of the wound at this stage of the operation, using double width Michel clips, about six to eight on the upper and lower edges of the wound. Bulky instruments are thereby avoided.

Sometimes a transverse incision may give excellent access to portal vein and inferior vena cava, as shown in Fig. 94. The man illustrated had mild cirrhosis and at the beginning of the operation lipo renal anastomosis was intended. However an excellent portal vein was disclosed on venography and a porta-caval anastomosis was done instead.

The next step in the operation is the dissection of the inferior vena cava. The peritoneum over the upper pole of the right kidney is divided, working upwards and inwards so as to arrive over the inferior vena cava where it constitutes the posterior border of the foramen of Winslow. This peritoneum is often excessively vascular, and it is wise to begin this incision by plunging the points of the haemostatic forceps (Roberts' are very suitable) through the peritoneum and dissecting the deeper structures away from the posterior surface of that layer before applying the clamps. The vena cava itself must be demonstrated before the peritoneum over its anterior surface is incised. Small gauze pellets mounted on long straight haemostatic forceps are very helpful in carrying out all dissections of vessels. Clamps on the cut edges of the peritoneum are tied off with thread. It is not safe to use diathermy for this purpose. The inferior vena cava itself is then freed by gauze pellet and scissor dissection. The vein is mobilised up behind the caudate lobe of the liver and it is here that care must be taken not to tear the small veins which pass from the liver or the capsule of the liver directly into the inferior vena cava. If the caudate lobe is thin and flat it may be found necessary to split it to give a better access to the upper reaches of the abdominal inferior vena cava. The vein is similarly dissected downwards to behind the pancreas and backwards to both renal veins, exposing about two and a half or three inches of its anterior aspect. The wall of the vena cava should then be cleaned scrupulously of all rough adventitial fibres. Tags of peritoneum which may

intrude during the construction of the anastomosis are removed and haemostasis secured

The portal vein itself is then approached. The venogram will have shown the position and state of the vein and its main branches. Further information about it can be obtained by very careful palpation of the gastrohepatic omentum between finger and thumb. The portal vein gives a feeling of 'bounce' or elasticity in an otherwise turgid and thickened structure. Its relation to the hepatic artery or its left and right branches should also be discovered before the dissection is proceeded with. The peritoneum is then incised over the portal vein posteriorly and within about one inch of the porta hepatis itself. By gauze pellet and scissor dissection the structures lying over the posterior aspect of the vein are penetrated and split aside and haemostasis is secured. In theory there should be no more than a layer of peritoneum and a few lymphatic channels, but in practice it is often found that the lymphatics have proliferated and hypertrophied into a body of considerable thickness and the peritoneum itself is often very vascular. The portal vein can usually be found without undue difficulty and the dissection around the vein is then proceeded with, the middle finger of the right hand often proving the most effective tool. The venogram should be available for frequent study if large branches have been seen entering the main trunk of the vein. As these are approached, great care must be taken to secure them between ligatures so as not to run the risk of serious haemorrhage. As the approach to the vein is opened up, the structures anterior to the portal vein, including the common bile duct, are retracted using a narrow malleable retractor hooked over at the tip. This can be adapted to suit the conditions of each case precisely and does not traumatise the tissues if correctly used. Gradually the portal vein is eased and completely separated from its bed right up to the inferior surface of the liver and down to the upper border of the pancreas. Tags of peritoneum and lymphatic tissue separating it from the inferior vena cava should be removed. In a kindly case this may be all the dissection that is necessary. Sometimes, however, the space between the inferior surface of the liver and the superior edge of the pancreas is not large enough for the portal vein to be approximated to the inferior vena cava without risk of angulation. Since it is necessary that the portal vein shall have a clean, smooth run in on to the stoma, it may be advisable to divide down into the upper edge of the pancreas itself so that there shall not be a rigid structure kinking the portal vein from below.

During the course of this dissection care must constantly be taken that no damage is done to the common bile duct and the hepatic artery or one of its main branches. Nothing should be divided or tied off until its exact nature has been determined. If the portal vein has been thrombosed it will

be more adherent and fixed and the dissection will be proportionately more difficult

A Blalock bulldog clamp is then applied to the lowermost intestinal end of the dissected segment of the portal vein and a heavy curved haemostat to the uppermost part in the porta hepatis. Moynihan forceps usually serve the purpose well. The portal vein is then divided as near to the Moynihan forceps as possible and the hepatic end of the vein tied off with a single heavy thread ligature.

If old thrombus is present it can be cut or scraped from inside the vein and leaves a surprisingly smooth intimal surface. For reaching the deeper extensions of the thrombus the Blalock clamp may have to be released and a Volkmann's spoon thrust up against the rush of blood and manipulated to remove clot as quickly as possible. Finger compression against the handle of the instrument will minimise blood loss.

The pressure of blood in the intestinal end of the vein may be so high that the Blalock clamp is blown off. This is a strangely unnerving sight when dealing with a very large vein in a large and powerful man. (A Crafoord cloth shod clamp is substituted for the Blalock and applied so that the handles protrude vertically from the wound.) The cut end of the portal vein is then approximated to the anterior surface of the inferior vena cava and the precise point of the anastomosis chosen. This is then picked up with large blunt-ended soft toothless dissecting forceps and a Tom Smith or other suitable clamp is applied to the inferior vena cava so as to pinch off a segment. The divided end of the portal vein is then further trimmed to give a clean cut and an even approximation if possible at an angle slightly less than a right angle. A small ellipse is cut away from the isolated segment of inferior vena cava using sharply curved Prince's scissors. The size of the hole is accurately adapted to the size of the portal vein. Both open veins are then well irrigated with citrate or heparin solution.

Four holding (or spacing) sutures of 000000 silk are then inserted one at the upper and one at the lower end of the anastomosis picking up vena cava and portal vein one on the anterior cut edge of the portal vein to hold it forwards and one on the right edge of the cut inferior vena cava to hold it outwards during the construction of the stoma. These spacing stitches are held with mosquito forceps.

A running stitch of waterproofed 0000 black silk (obtainable from Ethicon Sutures Ltd of Edinburgh) is then inserted beginning at the deepest point of the anastomosis as shown in Fig 95A. The suture is twenty inches long with a fine half circle atraumatic artery needle mounted at each end. An everting stitch of the reversed Connell type (or loop-on-adventitia) is used. Each needle puncture is made about 1 mm from its neighbour and $1\frac{1}{2}$ mm

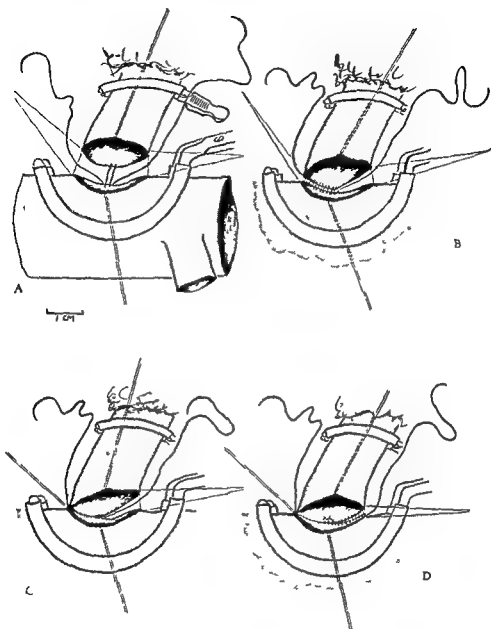


FIG 95

Four diagrams to illustrate the technique of end to side porta-caval anastomosis showing the insertion of the everting suture over the deeper half

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from the cut edges of the veins. Beginning from the deepest point, the anastomosis is made in four separate quadrants. In the first the stitch is worked upwards until the upper spacing stitch is reached. It is usually best to keep it pulled up after each square stitch has been inserted. When the quadrant has been completed the stitch is pulled up tight, the spacing stitch is tied, and the running stitch is tied to it (Fig 95B and C). The quadrant from the deepest point to the lower pole is then similarly completed (Fig 95D). The superficial half of the anastomosis is much easier as the needle can be passed into the inferior vena cava and out of the portal vein with one movement, whereas in the deeper quadrants each half of each stitch has to be done

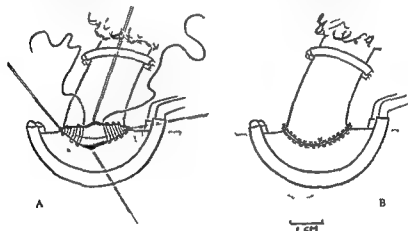


FIG 96

Two diagrams to show the completion of an end-to-side porta caval anastomosis

separately. As the two ends of the continuous suture approach the most superficial pole, the clamp on the portal vein is momentarily released in order to ensure that clots have not formed during the construction of the stoma. The clamp is immediately reapplied, the anastomosis once more washed with citrate and sucked dry. The last few stitches are inserted and the two halves of the superficial suture line are pulled up, counter traction being exerted on the spacing stitches at the upper and lower poles of the stoma. The ends are tied together tight enough to draw the edges of the anastomosis into accurate apposition, but not so tight that the suture line bunches up (Fig 96). All sutures are then cut short and the Tom Smith clamp is removed. If there is a leak it should be oversewn with a single square stitch. The Blalock clamp is then removed from the portal vein.

A representative fragment is removed from the edge of the liver for microscopical examination. It is wise to carry out this little manoeuvre between

interlocking catgut sutures so that there is no bleeding from the cut edge of the liver. The portal pressure is then taken again and the portal speed re-estimated. A stab drain is inserted just below the middle of the incision as shown in Fig 93, and the wound closed in layers. In cirrhotic patients the risk of disruption is greater than in almost any other condition, therefore the



FIG 97

Case No. 93. End to side porta caval anastomosis 20 mm in diameter. The everted suture line is well shown. (Mangot R. (1955) *Abdominal Operations* 3rd ed. New York: Appleton-Century-Croft Inc.)

abdominal closure must be strong. Interrupted far-and-near sutures of stainless steel wire standard wire gauge 33 have been found to be the most secure and the least liable to give trouble if the wound becomes infected.

Fig 97 shows that the anastomosis is in the nature of a flange joint. It does not contract nor does it thrombose if the portal vein is of good texture and not kinked or compressed. If the stoma is examined from within the abdominal cavity it is found to be of a natural shape and size (Fig 98).



FIG 98

Case No 159 Porta-caval anastomosis photographed from vena caval aspect
Lower picture trans illuminated to show the running everting suture
(Royal College of Surgeons Museum)

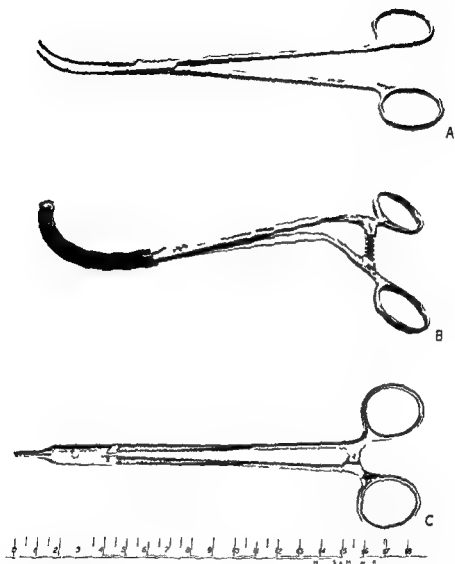


FIG. 99

- A Dissector/suture passer for the smaller radicles of portal and splenic veins etc
 B Modified Tom Smith clamp for the isolation of a segment of inferior vena cava
 C Special long needle holder with fine jaws for use with small curved and half-circle needles

APPENDIX 2

Fig 99 illustrates three special instruments which have been found very valuable in operations such as porta-caval anastomosis. Part A shows a fine long dissector/suture passer designed to help in the freeing of the smaller branches of the portal and splenic veins and inferior vena cava and in the passage of the sutures around them so that they may be divided between ligatures minimising the loss of blood during dissections. B illustrates a rubber-covered Tom Smith clamp which has been modified so that the handles come up out of the deep wound. Many other ingenious instruments have been devised such as the Barlow clamp but my preference remains for the ancient Tom Smith clamp which is so soft that it has never been known to do any damage to the intima of the inferior vena cava. A recent article by Henson and Rob emphasises the danger of rough handling of vessels in the course of vascular anastomoses leading to thrombosis. Fig 99c illustrates a long fine needle holder which has been devised for use with fine curved artery needles. (These instruments are obtainable from Down Bros and Mayer & Phelps of London.)

APPENDIX 3

LIENO-RENAL ANASTOMOSIS

The abdominal part of the incision extends from the umbilicus to the tip of the tenth costal cartilage on the left. It corresponds to the initial incision for porta-caval anastomosis (Fig 100). When the preliminary investigations and the examination of the relevant viscera have been completed and

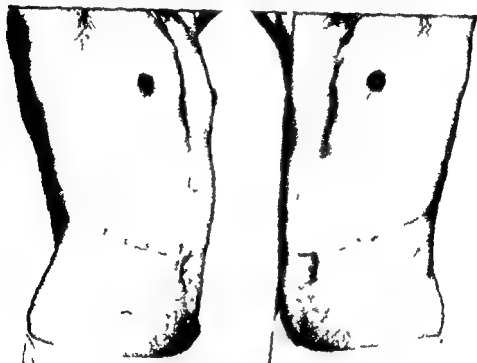


FIG 100

Case No 129 Incision begun for porta-caval converted into one for lieno renal anastomosis. Previous incisions for appendix abscess and exploratory laparotomy also shown.

the decision has been made to proceed with the anastomosis the incision is extended outwards and slightly upwards in the line of the tenth rib. Skin towels are affixed with double width Michel clips. The rib is resected the pleura opened and the diaphragm incised to give access to the lateral and posterior aspects of the spleen (Carter, 1947). Absolute haemostasis is secured. The cut edge of the diaphragm is then sewn with a running stitch of No 2 catgut to the cut edge of the muscles of the thoracic wall as the anaesthetist expands the lung. This closes the pleura, shuts off the lung from the field of operation and prevents blood accumulating in the recesses of the wound.

The spleen is then mobilised. It is occasionally possible to ligate the splenic artery as a preliminary step approaching it from the front through the divided gastrosplenic ligament. The outer layer of the lienorenal ligament is then divided between clamps. These are tied off with thread. The cut in the ligament is carried round to the upper part of the gastrosplenic ligament so that the upper pole of the spleen can be drawn downwards and medially. The splenic pedicle is then approached from behind and the artery secured if it has not already been tied from the front. It needs to be tied gently to avoid the risk of cutting through the wall of the artery. An aneurysmal tie should be used. The splenic vein is found and its relation to the pancreas discovered; the remaining attachments of the spleen are divided, preserving the splenic vein to allow drainage of blood into the body. Finally the splenic vein is clamped as near the spleen as possible and the spleen cut away.

The dissection of the splenic vein follows. This may be difficult or easy depending upon how closely it is enveloped with pancreas. The small pancreatic veins must each be divided between ties; the dissector/suture passer proving of value. The dissection will be more difficult if thrombosis has occurred. It is necessary to remember during these operations that a successful anastomosis may provide the one chance of normal existence for a young child. Patient dissection may be required to find a serviceable segment of vein which should measure 4 to 5 cm in length. After cleaning the proximal end is clamped with a Blalock or Blakemore clamp and the vein is citrated. The end is prepared for anastomosis with a clean cut and laid aside under a citrate pack.

The left kidney and renal vein are then mobilised and the pedicle cleaned to expose a 3 cm length of renal vein. All sympathetic fibres and rough adventitia around it are removed. A large suprarenal vein usually needs to be divided and the left spermatic or ovarian vein exposed. A short length of renal artery is cleaned and the kidney returned to its bed. The splenic vein is then brought down to the renal vein and a point chosen for the anastomosis. Any kinking is corrected by further dissection. The splenic vein should finally lie in a smooth quarter circle without redundancy.

The anastomosis end to side of splenic into renal vein is then made. Blalock bulldog clamps are applied (a) to the renal artery (b) to the distal end of the renal vein (if it branches early two separate clamps may be used one for each tributary) (c) to the proximal renal vein and (d) occasionally to the ovarian or testicular vein. The renal vein is then hemitranssected at the point selected for anastomosis and washed out with citrate (Fig. 101). Spacing sutures of 000000 silk are then inserted at the ends of this cut and in the outer lips of splenic and renal veins. The anastomosis using 0000 waterproofed silk is then begun at the most medial point of the stoma and

PORTAL HYPERTENSION

done in four quadrants, as for porta-caval anastomosis. If the veins are not of exactly the same size, slightly larger stitches will need to be taken in the larger vein so that the surplus is evenly distributed over the whole stoma.

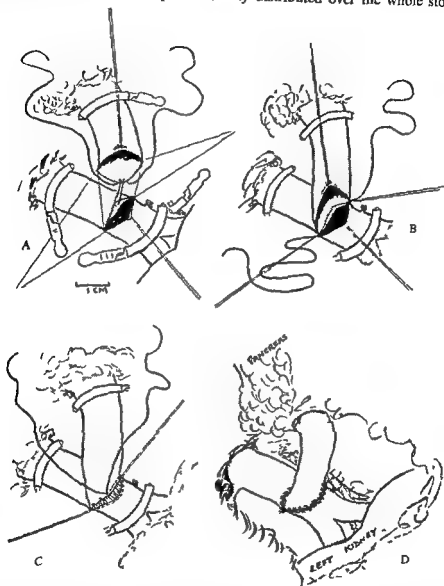


FIG. 101

Four diagrams to illustrate the technique of hepato renal anastomosis end to side

The anastomosis usually lies like an inverted T, as shown in Figs 101b and 102. Sometimes the hemitranssection of the renal vein allows the distal part to angulate backwards, and the splenic vein will then come in as one branch of a Y, an arrangement which appears very satisfactory.

APPENDIX 3



Fig 102

Case No 248 Leno-renal anastomosis of 15 mm diameter. From right to left can be seen lung divided diaphragm kidney and splenic vein coming down to join the renal by end to side anastomosis



Fig 103

Case No 36 Intravenous pyelogram before and after Leno-renal anastomosis to show that the kidney continues to function well (At postmortem some months later the anastomosis was functioning well)

PORTAL HYPERTENSION

The anastomosis takes about half an hour to make. If it is taking longer, the distal clamp on the renal vein and that on the renal artery should be removed for a moment to allow the kidney to be flushed through with arterial blood before the clamps are reapplied and the stoma completed. Fig 103 illustrates that a successful lienorenal anastomosis does not interfere with renal function.

Sometimes the renal vein is so large that it is possible to isolate a segment with a Barlow clamp and make the anastomosis in the manner of a porta caval with a longitudinal cut in the renal vein. The results, however, are not so satisfactory as when the vein is hemitransected.

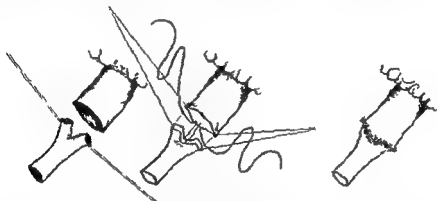


FIG 104

Method of anastomosing vessels of unequal size if a bifurcation is available
e.g. anastomosis between renal artery and portal vein

When the stoma has been completed the clamps are removed in the following order: (a) from proximal renal vein, (b) from distal renal vein (and from the ovarian if present), (c) from renal artery and (d) from the splenic vein. In size the stoma will be between 10 and 18 mm in diameter depending upon the age of the patient. The smaller the vein the less satisfactory the stoma. Finally the kidney is allowed to fall back into its bed and the position of the splenic vein adjusted.

If abnormal anteriorly placed renal arteries prevent the construction of an end to side anastomosis, the kidney should be sacrificed and an end to end junction made. This is the easiest of all shunt operations. If the splenic and renal veins differ greatly in size accurate coaptation can be obtained by incising into the bifurcation of the smaller vein as shown in Fig 104.

The temporary suture between the diaphragm and the thoracic muscles is removed and a stab drain is inserted just below the middle of the wound. The diaphragm is closed, the lung is expanded, and the chest and abdomen are closed in the usual manner. Intercostal drainage is not necessary.

APPENDIX 4A

PROXIMAL GASTRIC TRANSECTION AND RESECTION

The patient is placed on the operating table with the thorax rotated slightly to the right as shown in Fig 105. The incision is made along the line of the eighth rib from the angle to the costal margin (Fig 106). The thorax is opened and the diaphragm divided from costal margin to oesophageal hiatus. Haemostasis is secured. The inferior pulmonary ligament is then divided and the opening in the mediastinal pleura overlying the oesophagus is further enlarged so that the oesophagus can be isolated from its bed. All



FIG 105

Case No 247. Position for trans-thoracic oesophagotomy and proximal gastric resection. Chest rotated more to the right than the pelvis. Incision along the line of the eighth rib and costal cartilage.

vascular communications between it and the mediastinum are divided between ligatures or between clamps and as many mediastinal varices as possible are tied off or removed. The oesophagus is stripped as high as possible up to near the arch of the aorta. The dissection is then carried downwards into the abdomen through the divided diaphragm first along the greater curvature of the stomach and then down the lesser curvature to secure the left gastric pedicle. This operation would normally be simple but in cases of portal hypertension it can be exceptionally difficult. Matted brittle vascular adhesions containing hypertensive venous blood have to be divided and there is no

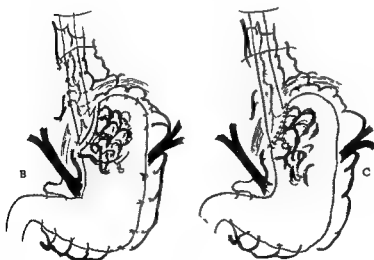


FIG 106

Case No 59 To show scar of neonatal umbilical and falciform ligament abscess and incision for (1) lino renal anastomosis along tenth rib to umbilicus and (2) proximal gastric resection along eighth rib



*Schematic drawing of VENOUS CONNECTIONS
of esophagus and proximal stomach*



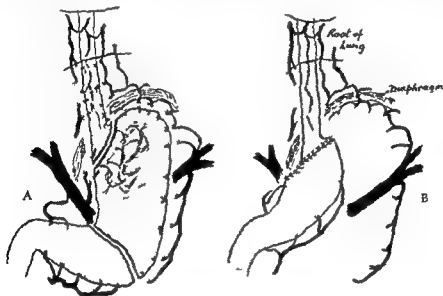
Isolation of the esophagus and proximal stomach, TRANSECTIONS and resuture

FIG 107

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short cut to simplicity. The dissection is a slogging match which makes some porta-caval anastomoses appear comparatively easy.

Fig 107A illustrates the venous connections of the stomach that have to be divided before a proximal gastric 'interruption' operation can be carried out. Figs 107B and C illustrate the operation of proximal gastric transection (Tanner). It will be seen that the gastric varices are not affected, and it is from these that subsequent haemorrhages are liable to occur, if indeed the varices do not reform in the oesophagus itself.



Isolation of distal oesophagus, RESECTION of proximal stomach and anastomosis

FIG 108

Figs 108A and B illustrate the operation of proximal gastric resection (Phemister and Humphreys). The varices at the cardiac end of the stomach are removed and the pyloric end is anastomosed to the cardia. It is wise in doing these operations to secure absolute haemostasis before beginning the anastomosis. All clamps are removed and every bleeding point is tied off. The anastomosis itself must be done with the most scrupulous attention to detail because of the liability to stricture formation. During the construction of the anastomosis a soft rubber Ryle's tube is passed down into the stomach so that this can be aspirated during the immediate post-operative phase and so that the patient can be fed through it from the third to the fifth day. An intercostal drain is inserted in the ninth interspace and attached to an under water seal before the thoracic wound is closed, the lung being expanded by the anaesthetist.

APPENDIX 4b

OPERATION OF OESOPHAGOTOMY AND LIGATURE OF OESOPHAGEAL VARICES

(George Crile Jun)

The position of the patient and the incision through the bed of the eighth rib is the same as for proximal gastric transection and resection. The inferior pulmonary ligament is divided and the mediastinal pleura incised. The oesophagus is isolated from the mediastinum and from its surrounding venous channels as shown in Fig 109A. A rubber tube is placed around it in order to give traction on the oesophagus and enable it to be pulled up slightly from the hiatus in the diaphragm. A soft rubber-covered clamp (Tom Smith's) is then applied across the lowermost point of the thoracic oesophagus. This gives the surgeon control of haemorrhage from the oesophagus as shown in Fig 109b. The oesophagus is then incised longitudinally (Fig 109c) and a continuous stitch of 0 or 1 chromic catgut is inserted to underrun the most prominent column of varicosities. If there is profuse bleeding within the oesophagus the Tom Smith clamp is retained during the earlier part of this procedure. If the bleeding is negligible the clamp is removed in order to give better access to the column of veins. This running stitch is inserted in an over and-over manner down to and beyond the lower end of the oesophagus. More and more mucous membrane is pulled up as the stitch is continued until gastric mucous membrane is encountered (Fig 109d). The stitch is then tied off and the other two main columns of oesophageal varices are similarly treated. A Ryle's tube is then passed down into the stomach and the oesophagus is closed in two layers over it. There will be only just enough room for the Ryle's tube and yet a barium swallow a fortnight later will show that no material constriction occurs.

The operation of oesophagotomy and ligature of oesophageal varices is designed to stop oesophageal haemorrhage and is no more than a temporary measure. The perioesophageal varicosities are therefore not tied off. In the operations of proximal gastric transection or resection on the other hand as many of the mediastinal varices as possible are ligatured and removed in order to prevent reflux of high pressure portal blood into the oesophagus above the transection.

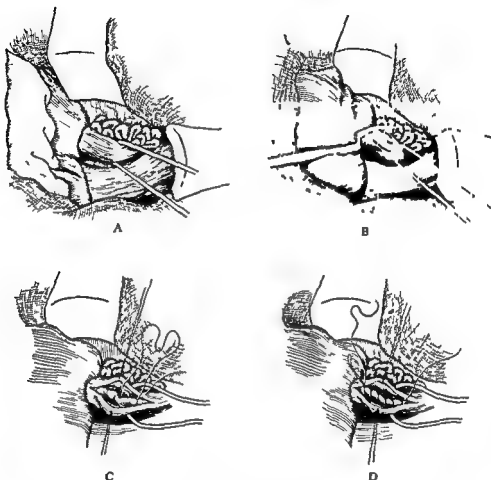


FIG 109

Case No 247 Four diagrams to illustrate the operation of oesophagotomy and ligation of oesophageal varices. To show isolation of the oesophagus from the varicose mediastinum with the aid of soft traction the precautionary application of Tom Smith's clamp to stop undue haemorrhage the opening into the lower oesophagus and the under running of the varices with a continuous catgut stitch (See Fig 43)

APPENDIX 5

SELECTED CASE HISTORIES

Case reports constitute the framework of any clinical research and in the following pages a number of case histories have been summarised to give a backbone to this Contribution to the Study of Portal Hypertension. It is hoped that the bones have not been picked too bare in eliminating all but relevant and essential details. Much of interest has been omitted and no idea can possibly be given in these brief statements of the diagnostic difficulties that many of these patients have caused. The excitement of accurate diagnosis and elucidation is necessarily absent.

The method of selection has been simple: The story of every patient who provides an illustration has been included with a few besides who have supplied some fact of interest or emphasised some point of importance.

The statement finishes at the end of 1956 only because this was the limit for the Jacksonian Prize Essay. New associations and new enthusiasms will write new chapters. Many of the conclusions are not final and the work continues.

Case No 1 I H FEMALE AGED 17 Congenital portal vein obliteration — Had had haemorrhages for two years. A very complete examination disclosed that she had Banti's syndrome the spleen being enlarged to two finger breadths below the costal margin and there being very large oesophageal varices on barium swallow. February 1948 operation: ilio renal anastomosis end to end using the Blakemore Lord tube.

Eighteen months later she had two haemorrhages and a third in July 1950. Haemoglobin 30 per cent. Second operation August 1950: deportalisation and sleeve resection of stomach through an abdominal incision. She made an un interrupted recovery.

1951 two more haemorrhages. 1952 further haemorrhage which required transfusion of two pints of blood. June 1952 third operation: anastomosis between superior mesenteric vein and the inferior vena cava. Portal venous pressure before anastomoses 370 mm water after 240 mm. Stoma measured 18 x 5 mm. She made a good recovery from this operation. During the past four and a half years she has remained in fair health the haemoglobin fluctuating between 48 per cent and 80 per cent. There have been two occasions only on which melaena stools have been passed and the anaemia has usually been attributable to an imperceptible ooze of blood into the alimentary canal. She remains at work and no further operative treatment is contemplated at present. It is presumed that the stoma has thrombosed.

PORTAL HYPERTENSION

Case No 5 L G FEMALE AGED 36 Advanced cirrhosis with gross ascites, rheumatic heart disease (mitral, aortic, and pulmonary stenosis) with auricular fibrillation—The patient became completely bedridden and made no progress under medical treatment. The liver and spleen were both enlarged one hand breadth below the right and left costal margins. Liver function poor as illustrated by albumin/globulin ratio of 2.4/2.4. In view of the hopelessness of the case it was decided that porta caval anastomosis should be attempted. This operation was done in March 1949 the anastomosis being done 'in continuity'. The patient developed hepatic coma post operatively but recovered consciousness on the twelfth day. She then developed bronchopneumonia from which she died on the twenty seventh post operative day. The encouraging feature of the case was that she did not form any ascitic fluid after the porta caval anastomosis. Fig 82 p 130 illustrates the anastomosis as found at postmortem. The hepatic part of the portal vein had thrombosed.

Case No 6 S G MALE AGED 25 Congenital portal vein obliteration—Admitted a fortnight after the onset of a massive haematemesis requiring the transfusion of 18 pints of blood and 3 pints of plasma. Haemoglobin 62 per cent. Liver function tests normal. The spleen had been removed fourteen years previously on account of Banti's syndrome. Operation in February 1949: anastomosis between a large cavernomatous channel and the inferior vena cava. Stoma approximately 12 mm in diameter.

Four months later he had another massive haemorrhage. This required the transfusion of a total of 26 pints of blood. In June 1949 the proximal two thirds of the stomach and the distal two inches of the oesophagus were resected and the oesophagus was anastomosed to the pyloric end of the stomach. From this operation he made an uninterrupted recovery and has been free from haemorrhage since. He does heavy work without difficulty and rarely suffers from dyspepsia.

In 1952 he had a hernia repaired and the opportunity was taken of carrying out oesophagoscopy: no varices were seen and the junction between oesophagus and stomach was healthy. Portal venous pressure was 460 mm water.

The operation specimen of this man's stomach is illustrated in Fig 41.

Case No 7 H G MALE AGED 51 Moderate cirrhosis due to chronic arsenicalism.—For five and a half years he had been in the habit of taking a tonic which contained 5 minims of liquor arsenicalis in each dose. He had usually had three doses a day. Five months before admission his abdomen had begun to swell and altogether more than 50 pints of ascitic fluid had been withdrawn. The ascites persisted in spite of full medical treatment. On examination his abdomen was grossly distended with ascitic fluid and he showed marked keratosis punctata and the raindrop pigmentation of chronic arsenicalism. The spleen was enlarged to percussion. The blood picture was normal and his liver function tests were fair as illustrated by albumin/globulin ratio of 4.6/1.4. Oesophagoscopy showed that there were small varices present.

Medical treatment was not producing any benefit so porta-caval anastomosis was done in March 1949. The anastomosis was constructed 'in continuity'. The

APPENDIX 5

patient made an uninterrupted recovery and from the moment of operation formed no more ascitic fluid. He returned to work and continued in good health until 1954 when a carcinoma of the bronchus was discovered following a haemoptysis. The affected lung was resected by Mr O S Tubbs but metastases had developed from which he died in August 1955 six and a half years after porta caval anastomosis. This was seen at postmortem to be in a perfectly satisfactory state with neither end thrombosed.

Case No 13 H B MALE AGED 45 Moderately advanced cirrhosis following infective hepatitis seventeen years previously.—Four months before admission vomited 2 pints of blood. Following this the abdomen became distended. The ascites improved with medical treatment and on abdominal examination an enlarged spleen and a hard nodular liver could be felt. Liver function considerably deranged as indicated by thymol turbidity of 15.5 units and serum proteins albumin/globulin 3.5/3.2. Blood count: haemoglobin 72 per cent WBC 4400 platelets 70000. Size of spleen estimated at 10 x 5 x 4 inches. December 1949 lieno renal anastomosis. Portal venous pressure reduced from 245 to 185 mm water. Diameter of stoma only 12 mm. Post operative course satisfactory for two days and then patient developed hepatic coma. Replacement transfusion of two pints of blood given without change. Became jaundiced. Five days after lieno renal anastomosis when the patient was undoubtedly dying the liver was arterialised by anastomosing the right renal artery to the hepatic end of the portal vein. He died twelve hours after this second operation. Postmortem both stomata were found to be in a satisfactory condition. (For 'arterialisation of liver' see page 108).

Case No 15 C W MALE AGED 33 Mild cirrhosis of the idiopathic type.—During 1948 and 1949 had three severe intestinal haemorrhages. On examination there was little evidence of cirrhosis but the spleen was enlarged three finger breadths below the costal margin. Liver function tests were within normal limits. February 1950 oesophagoscopy showed varices gastroscopy a congested gastric mucous membrane. The patient passed a large melaena stool on the following day. One week later lieno renal anastomosis was done the portal venous pressure being reduced from 435 mm to 285 mm water. The stoma measured 20 x 10 mm. Post operatively the patient's condition gave considerable cause for anxiety on account of persistent abdominal distension and pyrexia. Twice in March the left subphrenic and left perirenal spaces were explored for possible abscesses but no pus was found. The pyrexia gradually subsided and subsequent events beginning with a haemorrhage two years later suggested that there was considerable splenic and mesenteric thrombosis following the first operation. He continued in fair health till 1954 four years after the lieno renal anastomosis when further haemorrhage compelled a review of his case. Porta caval anastomosis was done. Since then his condition has been satisfactory but he has not been able to return to heavy work.

PORTAL HYPERTENSION

Case No 21. W C., MALE, AGED 29 Presumed extrahepatic portal vein obliteration.—A case of Banti's syndrome fully investigated in 1948. liver function tests normal, liver biopsy, histological features normal. The patient died of his second haemorrhage in April 1949 before medical aid could be summoned

Case No 24 V S., FEMALE AGED 30 Moderate cirrhosis following toxæmia of pregnancy.—Baby delivered eighteen months before admission. A further pregnancy was accompanied by symptoms and signs of advanced cirrhosis. Hysterotomy was carried out in July 1949. At that time there was serious derangement of liver function as indicated by the serum proteins albumin/globulin ratio 2.4/5.2. Oesophageal varices were demonstrated on oesophagoscopy. Medical treatment produced marked improvement. She returned to full work. In August 1953 she had a massive haematemesis and in February 1954 another haemorrhage which necessitated porta-caval anastomosis. This was done in March 1954. She made a good recovery and again returned to full work. Liver function tests were satisfactory

In July 1955 an attack of appendicitis necessitated further operation. When the appendix was removed the opportunity was taken of repeating the portal venogram which is shown in Fig 78. It illustrates a well functioning porta caval anastomosis with full decompression of the portal tree

Case No 36. E G., FEMALE, AGED 48 Idiopathic cirrhosis associated with retinitis pigmentosa—1948 splenic anaemia. 1949 haematemesis requiring transfusion of 2 pints of blood. 1950 severe haematemesis, transfusion of 7 pints. Diagnosis of moderate cirrhosis with Banti's syndrome was confirmed. October 1950 lieno-renal anastomosis. Two months later the patient became acutely ill jaundiced, and comatose. She died within three days of the onset of this attack, and at postmortem the liver, which was small, smooth, and fibrotic, showed acute, diffuse hepatic necrosis (Fig 103)

Case No 40 M A., MALE AGED 6 Congenital atresia of the common bile duct with secondary biliary cirrhosis and portal hypertension; gastric ulcer; hepatoma.—He was jaundiced at birth and this persisted with remissions until he died. His liver and spleen were both noticed to be hard and enlarged four finger-breadths below the costal margins at the age of 4 when he passed a melaena stool. Soon after that he developed ascites and telangiectases. When 5 years old he had a haematemesis. His condition slowly and steadily deteriorated. At the age of 6½ in October 1950 the liver function was very poor—serum proteins albumin/globulin 2.2/3.0. It was hoped that by intensive medical therapy his condition might be improved so that an exploratory operation could be done. The intention was, if possible to relieve the biliary obstruction and at a later date the portal hypertension. However, he started to bleed, became comatose, and died. At post-mortem the liver was shown to be extremely fibrotic and containing a large hepatoma (see Fig 28). The diagnosis of congenital biliary atresia was confirmed and it was found that the fatal haematemesis had come from a large vessel eroded in the base of a gastric ulcer

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Case No 42 B C, MALE, AGED 44 Idiopathic cirrhosis, severe.—Suffered from gross ascites which had begun seven months previously. He was very ill. Paracentesis abdominis of 18 litres improved his condition (see Fig 67). Barium swallow showed that gastric varices were present, serum proteins: albumin/globulin 3.2/2.3



FIG 110

Case No 42 A Renal portal anastomosis. **B** Porta renal anastomosis after both had been opened out postmortem seen from the intimal aspect

His severe illness, rapid deterioration, and failure to respond to medical treatment suggested that his only hope was arterialisation of the liver. In November 1950, therefore, a double anastomosis was constructed, portal vein anastomosed to the stump of the right renal vein after the kidney had been removed, and the right renal artery anastomosed to the hepatic end of the portal vein. Heparin was administered post-operatively and unfortunately he had a large retroperitoneal haemorrhage on the day after operation and died in a state of coma. At postmortem the anastomoses were in a satisfactory condition, as shown by the accompanying illustration, Fig 110. The diagnosis of advanced multilobular cirrhosis was confirmed.

Case No 47. A A., MALE, AGED 58. Extrahepatic portal obstruction due to compression by a hypernephroma of the right kidney which contained a large cirroid

PORTAL HYPERTENSION

aneurysm—Five years before admission he had had a car injury followed by a melaena. He was treated for gastric ulcer. He had had his first haematemesis two years before admission when it was discovered that he had a loud 'machine' murmur over the right lower chest and liver area. His blood pressure was about 230/120 mm mercury. Liver function tests were normal. Barium swallow showed the presence of small oesophageal varices and an intravenous pyelogram showed that the right kidney was displaced outwards and upwards. As a result of pre-operative discussions it was postulated that an arteriovenous fistula and a tumour would both have to be present in order to account for all the physical signs. In March 1951 the region of the right kidney was explored through a horizontal incision and a hypernephroma was found to be compressing and displacing the portal vein. It arose in the upper pole of the kidney which contained a very large arteriovenous curdoid aneurysm. The renal artery was tied and then the renal vein and the right kidney was removed. On opening the specimen it was found that the tumour protruded into the enlarged renal vein like the tip of a finger.

The patient made a very satisfactory recovery but the blood pressure altered little. He developed a duodenal ulcer which was treated by partial gastrectomy later in 1951. Since then he has had a stroke from which he has partially recovered and two attacks of subacute intestinal obstruction. He is an invalid on account of his cardiovascular state and partial paralysis but there has been no recurrence of the hypernephroma.

Case No 49 S J FEMALE AGED 10 Congenital portal vein obliteration—At the age of 8 she had a severe haematemesis associated with Banti's syndrome for which splenectomy had been done. Haematemeses had continued. On examination her general condition was excellent. Liver function tests were normal. Oesophagoscopy showed numerous large varices extending up to 22 cm from the upper incisors. In March 1951 a 'makeshift' porta caval anastomosis was constructed between a large cavernomatous channel and the inferior vena cava. The portal venous pressure was reduced by only 55 mm water from 300 to 245 mm. Haemorrhage from the porta hepatis was difficult to control during this operation and required the ligation of a number of large vessels. Post operatively she became jaundiced and developed a biliary fistula. The fistula healed and the jaundice fluctuated suggesting partial obstruction to the biliary passages.

Five months later the wound was re-explored and the obstruction was found to be to the common hepatic duct. A loop of jejunum was brought up and anastomosed in Y to the duct above the stricture. The portal venous pressure was 365 mm. water and a venogram showed that the porta caval anastomosis had become obliterated.

The jaundice cleared and the patient has led a normal life since the second operation though there has been one minor haemorrhage.

Case No 51 I P FEMALE AGED 43 Advanced cirrhosis of the idiopathic type—Two years before admission massive haematemesis from oesophageal varices. She then had four operations done elsewhere—proximal gastric transection, ligation

APPENDIX 5

of mediastinal veins anastomosis of inferior mesenteric vein to inferior vena cava and attempted porta caval anastomosis. The haemorrhages persisted and the condition became desperate. Liver function tests indicated considerable derangement



FIG. 111

Postmortem venogram of Case No. 51 (see text). The line of previous gastric transection lies between the two white arrows.

serum proteins albumin/globulin 3.4/3.1 thymol turbidity 14 units. In May 1951 the portal vein was re-explored and in the end of a very difficult operation a satisfactory porta caval anastomosis was constructed. The patient died post-operatively

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At postmortem the diagnosis was confirmed. Before removing any organs however the portal vein was clamped and the portal tree injected with a suspension of bismuth and gelatine. An X ray was taken and the appearances are shown in Fig 111. An analysis of the rather confused picture shows that the blood flows back from the splenic vein along the short gastric veins to the gastric varices and so down to the line of transection which runs between the two white arrows. The effect of the first operation was therefore the opposite of what was intended in that the blood appears to be held up in the proximal part of the stomach rather than held down and prevented from reaching the varices.

Case No 53 E G MALE AGED 49 Biliary cirrhosis secondary to stricture of common bile duct following cholecystectomy nineteen years previously—Complaint persistent jaundice and recurrent haematemesis. Liver and spleen were both palpable. oesophageal varices were present and the liver function was moderate. serum proteins albumin/globulin 4.00/2.75 serum bilirubin 8 mg/100 ml. In April 1951 a biliary stricture was excised and calculi were removed from the common hepatic duct which was reconstructed over a T tube. The jaundice cleared and the patient's general condition improved in spite of recurrent haematemesis. In August 1953 he developed ascites following a severe haemorrhage. After a course of medical treatment his liver function appeared reasonably good. serum proteins albumin/globulin 3.7/3.1 and it was decided that an operation for the relief of portal hypertension was imperative. In October an end to end ilio renal anastomosis of 13 mm diameter was constructed. The portal venous pressure was reduced from 345 mm to 230 mm water. The thrombosed condition of his portal vein at this operation is illustrated in Fig 17 p 21.

Following this operation the patient made a good recovery. The ascites disappeared and he returned to work as a pig and cattle farmer.

In February 1954 he was re-admitted as an acute emergency with intestinal obstruction. This was treated conservatively in the belief that it was due to a mesenteric thrombosis. The patient made a good recovery and again returned to work.

Early in 1955 he developed a severe ascending cholangitis from which he died.

Case No 59 M H MALE AGED 6: Congenital portal vein obliteration associated with suppurative omphalitis (see Fig 106) Haematemesis and melæna from Banti's syndrome. October 1951 ilio renal anastomosis followed by further haemorrhages. November 1954 transthoracic proximal gastric resection. The liver was re-examined at this operation and its normal state confirmed.

Since this second operation the patient has according to his father never been better though he is thin. He eats well though his meals are small and he leads a normal life for a schoolboy.

Case No 61 M B FEMALE AGED 57 Advanced idiopathic cirrhosis—In October 1951 she had been explored on account of unexplained ascites. A small nodular cirrhotic liver had been discovered. Function considerably deranged. serum

proteins albumin/globulin 3.2/2.9 Oesophageal varices were not demonstrated. A course of medical treatment was begun and continued intensively without relief of the ascites but with some improvement in the liver function (see Fig 69). December 1951 ligation of coeliac axis and hepatic artery (reduction in portal venous pressure from 260 to 140 mm water). Her post-operative course gave considerable cause for anxiety, but she disposed of her peripheral oedema with the aid of mersalyl injections. Albumin/globulin ratio five weeks after operation 3.8/2.2. She went home in February, free from ascites, and returned to full work as a hotel proprietress in May. She closed down at the end of the season in October and then had a severe haemorrhage with reduction of the haemoglobin to 21 per cent. She recovered and in January 1953 the serum proteins were albumin/globulin 4.25/2.63. She was still free from ascites and feeling very well. At Whitsun she again opened up her hotel and worked through until the end of July 1953 when she had another severe haematemesis from which she died.

Case No. 62 A J, MALE, AGED 51. Idiopathic cirrhosis of moderate extent.—He developed Banti's disease with haemorrhages at the age of 19. Splenectomy was done by Sir James Walton in 1921, using an incision which divided the costal margin at the tenth costal cartilage and extended down towards the umbilicus. He was free from haemorrhages for about five years, but since the age of 26 he had had a haematemesis every twelve to eighteen months. Recently they had been becoming more severe and keeping him off work for two to three months. In December 1951 porta caval anastomosis was attempted, but it was found that the portal vein was obliterated by clot. Dissection was continued, but unfortunately produced a massive haemorrhage which required packing. A biliary fistula developed which healed and the patient became jaundiced. June 1952 the jaundice persisted and the biliary fistula recurred. The operation of cholecystojejunostomy in Y was done with relief of jaundice and marked improvement in the patient's condition. Since then he has continued to have haemorrhages every nine to twelve months. During the latter part of 1956 his oesophageal varices have been injected with sclerosant solution on three occasions and the process is to be repeated during 1957.

Case No 63 R B, FEMALE AGED 5. Congenital portal vein obliteration.—Had three severe haemorrhages between the age of 3½ and 5. December 1951 end to side ilio-renal anastomosis. Only a small, poor segment of splenic vein could be found or demonstrated by venography (Fig 112, arrowed) (Hunt, *Proc. R.S.M.*, 1952). Haemorrhages continued. July 1952 'makeshift' porta-caval anastomosis, using a cavernomatous channel. Haemorrhages continued. September 1952 proximal gastric transection with stripping of the oesophagus through the bed of the left ninth rib. Following this operation the patient was satisfactory for thirteen months. She then had a severe cold followed by a melaena stool. Oesophageal varices were injected with a sclerosant in October 1953. Three days later she had a very severe haemorrhage which necessitated an emergency proximal gastrectomy. The bleeding came from gastric varices. The patient made an excellent recovery from this operation and

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returned to normal life. She did well at school for two years, but in 1956 she has had two further episodes of bleeding. Oesophagoscopy shows that she has no oesophageal varices but an erosive oesophagitis from which the blood oozes.



FIG 112

Portal venogram in a case of congenital portal vein obliteration to show no more than a small segment of poor splenic vein (arrowed)

Case No 65 C L, FEMALE AGED 13 Cirrhosis hepatis of moderate degree following infective hepatitis at the age of 9.—Severe haemorrhage two months before admission. November 1951 end-to-end ileo-renal anastomosis 8 mm diameter. The kidney had to be sacrificed because the renal artery was duplicated, both branches lying anteriorly to the renal vein. The portal venous pressure was reduced from 550 mm water to 290.

Her liver function tests improved, but in December 1952 she developed a curious 'adenoidal' facies. Her mental state deteriorated and in 1953 the diagnosis of hepatolenticular degeneration (Kinnear-Wilson's disease) was made. She died in March 1954 in a state of hyperpyrexia.

Case No 66 A B, MALE AGED 26 Idiopathic cirrhosis, mild.—At the age of 23 he had begun to have haematemesis and at 24 the spleen had been removed elsewhere. The appearances were typical of ■ Banti's splenomegaly. Haemorrhages had continued. In November 1951 end-to-side porta caval anastomosis was done, but only after a very difficult dissection and the removal of a large organised thrombus which occupied two thirds of its lumen. The stoma measured 15 mm in diameter and the portal venous pressure was reduced from 370 mm to 150 mm water. Twenty-four hours after operation a course of heparin was given which lasted for five days.

He made ■ satisfactory recovery but two months later had to be re-admitted with sub-acute intestinal obstruction, abdominal distension, and incipient hepatic

coma. The usual therapy for intestinal obstruction was given, short of operation, but his condition deteriorated and the coma deepened. Heparin was added to the treatment for forty eight hours with remarkable and satisfactory improvement. Since then his condition has been good and he has remained at heavy work (see Fig 14 p 18)

Case No 70 F D, MALE, AGED 26. Idiopathic cirrhosis, mild in nature.—Two severe haemorrhages during the nine months before admission. Had been serving in the Far East for seven years. On examination; palmar blush and a hard edge to the liver suggested cirrhosis, the spleen was very enlarged, extending down to below the level of the umbilicus. Oesophageal varices were present. A third haematemesis necessitated the transfusion of $5\frac{1}{2}$ pints of blood. January 1955 end-to side ileo renal anastomosis the stoma measuring 16 mm in diameter. The portal venous pressure was reduced from 500 mm to 160 mm water. The spleen measured $13 \times 7\frac{1}{2} \times 5$ inches. Since the operation he has led a normal life and remained in good health (see Fig 62, p 78)

Case No 71. F K, FEMALE, AGED 58. Idiopathic cirrhosis hepatitis of moderate extent.—She gave a seven month history of ascites. She had been explored elsewhere three months before admission when a cirrhotic liver and an enlarged spleen had been found. General condition was poor but the liver function tests were fair, as illustrated by the serum proteins albumin/globulin 3.7/3.3. May 1952 ligation of the common hepatic artery. The rate of formation of ascitic fluid increased post operatively from 200 cc a day to 310 cc a day. Over the years, however, the ascites has gradually absorbed and the most recent report in May 1956 shows that she now has no ascites and that her serum proteins are much improved, albumin/globulin 5.2/2.05

Case No 74 E A, FEMALE AGED 45. Extrahepatic portal obstruction due to compression of the portal vein by the displaced left kidney.—Her main complaint was of splenic anaemia, haemoglobin 54 per cent, WBC 1,600, platelets 50,000, reticulocytes 3.6 per cent. At exploration in March 1952 it was found that the enlarged spleen had pushed the left kidney over to the right where it had become wedged behind the foramen of Winslow. The portal vein was greatly compressed and the portal venous pressure was 400 mm water. The spleen was removed the kidney returned to its usual position, and the portal venous pressure dropped to 110 mm. Since then the patient has remained in excellent health.

Case No 75 N E, FEMALE AGED 43. Idiopathic cirrhosis of moderate extent.—For four and a half years she had suffered from abdominal distension, with enormous enlargement for the six weeks prior to admission. Liver function tests were reasonably satisfactory proteins albumin/globulin 3.5/2.6. In February 1952 the abdomen was explored the diagnosis confirmed and the coeliac axis ligated. This produced a drop in portal venous pressure from 360 mm to 330 mm water. The common hepatic artery was found to be still pulsating so it was then tied apart.

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from the coeliac axis and the pressure was reduced further to 285 mm water. The post operative course was not smooth on account of necrosis of the spleen which finally sloughed out leaving the patient with a persistent sinus. This was explored in May 1954 laid widely open and packed. After this the wound healed. The liver function tests had improved serum proteins albumin/globulin 5.4/2.5. The patient has remained free from ascites. She has a mild degree of hypochromic anaemia.

Case No 76 J S MALE AGED 36 Cirrhosis hepatis of moderate extent arising as a result of three years imprisonment in the Far East with frequent attacks of malarial dysentery wet and dry beri beri tropical ulcers and starvation oedema. Two and a half years before admission noticed undue fatigue and jaundice. He had severe intestinal haemorrhages on at least four occasions before admission. On examination he showed the stigmata of cirrhosis his liver was enlarged and hard and his spleen was palpable to below the level of the umbilicus. There was no ascitic fluid present. Liver function tests were satisfactory. March 1952 end to side lienorenal anastomosis. The portal venous pressure was reduced from 370 mm to 215 mm water. The original intention at this operation was to do a porta caval anastomosis but the veins of Retzius were so enormous (see Fig 33 p 43) that the lienorenal anastomosis was done so as to avoid the most vascular region of his retroperitoneal tissues.

Since operation his condition has been very satisfactory and he has continued at full work as a bartender to a golf club. He himself is teetotal.

Case No 78 W M MALE AGED 41 Idiopathic cirrhosis of moderate extent. —Three haematemeses during the five months before admission. On examination stigmata of cirrhosis were present (see Fig 63). The liver and spleen were both easily palpable. There was no ascites. April 1952 end to side porta caval anastomosis the stoma measuring 15 mm in diameter the portal venous pressure being reduced from 270 mm to 160 mm water. His post operative course was uneventful and he returned to work as a captain in the merchant navy. During 1954 however his condition began to deteriorate. He was invalided out of the service and died of an unknown cause in July 1955. It is presumed that his liver failed him.

Case No 80 W C MALE AGED 43 Arsenical cirrhosis of moderate extent. —For sixteen years this patient had had dermatitis herpetiformis which he had treated for thirteen years by taking Fowler's solution three times daily. During the eighteen months before admission he had had three haematemeses. The liver and spleen were both enlarged and he had oesophageal varices. April 1952 end to side porta caval anastomosis. Stoma 17.5 mm in diameter. Portal venous pressure rose from 265 mm to 300 mm water. Post operative course was satisfactory. His haemoglobin rose by nearly 20 per cent and his liver function improved as indicated by a serum albumin of 4 before operation rising to between 4.8 and 5.1 after porta



FIG 113

The cutaneous manifestations of chondrosarcoma. A and B show the lesions on the back and palm of the hand, respectively. C shows the lesions on the forearm. C also shows the curved white line of the chondrosarcoma.

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caval anastomosis. He returned to work as an accountant and obtained considerable promotion in his profession.

Fig 113 shows keratosis punctata of the palms and soles, early epithelioma of the forearm and the curved white nails of cirrhosis hepatis.

Case No 81 A W FEMALE AGED 5 Idiopathic cirrhosis of moderate extent.—Had been unwell with icterus or jaundice and occasional melæna stools from the age of 2½. On examination the patient was ill and anaemic, the spleen was grossly enlarged and a rounded tumour was palpable in the right upper quadrant of the abdomen. A provisional diagnosis of carcinoma of the liver was made. In June 1952 the abdomen was explored and the mass was found to be an extremely mobile cirrhotic liver (see Fig 25 p 30). The portal venous pressure was 430 mm water. An end to side porta-caval anastomosis of a diameter of 12 mm was constructed and the pressure dropped to 370 mm water. The patient made a good recovery from this operation.

Since then she has had seven haemorrhages of varied severity but has maintained good health and been able to lead an active life as a schoolgirl. Leno renal anastomosis is considered necessary but is being deferred until such a time as the patient's veins will be large enough to carry her through life. Her liver function remains good.

Case No 85 M C FEMALE AGED 36 Congenital portal vein obliteration.—At the age of 10 she began to have haemorrhages associated with a Banti's syndrome for which the spleen was removed at the age of 15. The haemorrhages continued and became more severe during the years preceding admission. September 1952 anastomosis of the inferior mesenteric vein to the inferior vena cava. Portal venography showed that the inferior mesenteric vein was the only reasonable venous channel remaining in the portal system (see Fig 9 p 14).

Soon after this operation she had another haemorrhage and in December 1952 a proximal gastric transection operation was done. After this she had no haemorrhage for eighteen months but then she began to bleed again and in April 1955 a proximal gastric resection was done. Since then she has had no further haemorrhage but has suffered from considerable dyspepsia with regurgitant oesophagitis.

Case No 89 C W FEMALE AGED 53 Advanced cirrhosis following infective hepatitis.—Complaint ascites which failed to respond to medical treatment. Gross derangement of liver function as illustrated by serum proteins albumin/globulin 3.1/4.4, pseudocholinesterase 29 units. November 1952 end to side porta-caval anastomosis which caused the portal venous pressure to drop from 295 mm. to 180 mm water. The portal vein contained old clot which had to be removed before the stoma could be constructed with satisfaction. Heparin was therefore administered post-operatively but this was accompanied by massive intra-oesophageal and retroperitoneal haemorrhage from which the patient died (see Fig 40 p 49).

Case No 90 T. H., MALE, AGED 2½. Congenital portal vein obliteration.—Patient complaining of recurrent oesophageal haemorrhages, beginning at the age of 10 months. The spleen was enlarged. There was no evidence of hepatic disease. It was decided that operation was imperative, but that the splenic vein should be spared for an effective operation on a later occasion. In November 1952, therefore, proximal gastric resection was carried out.

Since that time the patient has had no further haemorrhage, but a stricture developed at the suture line. This has required treatment by repeated dilatations. The last was done in January 1955. Since then the patient has been able to lead an almost normal life (see Fig 7, p 12, the venogram of a typical portal cavernoma).

Case No 91 S. McL., FEMALE, AGED 6. Congenital portal vein stricture (see Fig 10, p 14)—Seven months' history of four episodes of haemorrhages before admission. September 1952 end-to-side lieno renal anastomosis. Stoma measured 10 mm in diameter. Portal venous pressure dropped from 440 mm to 220 mm water. The patient regained excellent health and continued the normal activities of a schoolgirl for three years, when she had two haemorrhages in quick succession. In May 1955 end-to-side porta-caval anastomosis was done. Venogram showed the lieno renal anastomosis had become completely occluded, the collateral vessels around the foramen of Winslow had increased considerably, and the dissection of the portal vein was, therefore, much more difficult than anticipated. The liver again was quite normal. Portal venous pressure 280 mm water before, 225 mm after construction of the anastomosis. The speed of flow 6 cm per second before, 10 cm after. The patient made an uninterrupted recovery from the operation and has been leading a normal life since.

Case No 92 J. H., MALE, AGED 37. Cirrhosis of mild extent coming on twelve years after being in East Prussia as a prisoner-of-war.—He had been a prisoner for six years during which time he had had sundry dietary deficiencies and pulmonary tubercle. During the six months before admission he had had several severe intestinal haemorrhages. September 1952 end-to-side lieno renal anastomosis. The stoma measured 17 mm in diameter and the portal venous pressure was reduced from 235 mm to 105 mm water.

On the nineteenth post-operative day the patient became mentally deranged and passed into coma. This was treated with intravenous dextrose, plasma, aureomycin and heparin. He made a satisfactory recovery, but passed into coma again while at a convalescent home, and was admitted to a hospital where he died of acute hepatic failure.

Case No 93 E. W., FEMALE, AGED 63. Idiopathic cirrhosis of moderate extent.—Had had two haematemeses during the nine weeks before admission. The liver was enlarged and hard, palmar blush was present and she had a caput medusae. Liver function tests were satisfactory, albumin/globulin 5 1/2:8. October 1952 porta-caval anastomosis was done. The stoma measuring nearly 20 mm in diameter.

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(Fig 97 p 150) The portal venous pressure was reduced from 325 mm to 295 mm water. The patient made an uninterrupted recovery.

Two years and three months later she began to become slow and incoherent in her speech (see Fig 76 p 115). She became slow mentally and dropped into coma. She recovered from this and entered a semi-conscious state in which she remained for nearly two years before dying of liver failure at the age of 67. The serum albumin remained at a reasonable level throughout this time but the pseudocholinesterase fluctuated between 4 and 27 units. All forms of treatment for portal systemic encephalopathy were tried on this patient with little benefit.

Case No 95 J R FEMALE AGED 6 Idiopathic cirrhosis of moderate extent—This patient was investigated originally in 1951 on account of frequent nose bleeds and persistent anaemia. Her spleen was found to be moderately enlarged and she had the stigmata of cirrhosis (see Fig 61c p 77). Her health has fluctuated during the last five years. There was one serious setback when her liver function deteriorated her serum albumin dropping from 3.8 to 2.2 and her pseudocholinesterase from 33 to 26 units. She developed ascites which responded to medical treatment. However she made a good recovery from this attack and in September 1956 the albumin had risen to 3.7 and the pseudocholinesterase to 37 units. At present there is no accepted indication for surgical intervention and she remains under close medical supervision.

Case No 96 A V FEMALE AGED 67 Advanced idiopathic cirrhosis—Two months before admission haematemesis followed by ascites. Cholecystectomy and choledochotomy had been done five years and four years before and it is possible that her cirrhosis was partly due to a previous brief attack of obstructive jaundice. She improved with medical treatment serum proteins reaching albumin/globulin 3.8/2.8 pseudocholinesterase 39 units and haemoglobin 76 per cent but without relief of her ascites which required tapping. October 1952 exploratory operation with a view to porta caval anastomosis. This was abandoned because of the excessively dense and vascular adhesions resulting from the previous operations. In November an end to side ileo renal anastomosis was constructed the incision being extended outwards through the bed of the eleventh rib without opening the pleura. Recovery was very satisfactory with relief of ascites. The proteins fell to albumin/globulin 2.5/2.6 post operatively. The serum albumin has recovered to an average of 4.4 over the last four years (best 5.4) and the pseudocholinesterase to more than 60 units (best 77). Her general health is very good with a haemoglobin level usually of more than 100 per cent.

Case No 100 J W FEMALE AGED 51 Toxicopathic hepatitis due to carbon tetrachloride (and (*) inorganic arsenic)—In her work as an industrial chemist she had handled carbon tetrachloride for fifteen years. She took a blood tonic containing arsenic to neutralise the evil effects of the chemical. For fourteen months before admission she had had a number of haematemeses and melaena. Exploratory operation elsewhere had confirmed the diagnosis of cirrhosis and eliminated that of

ulcer. She was a fat woman and neither liver nor spleen was palpable. The only clinical evidence of cirrhosis was a mild palmar blush. The cutaneous manifestations of arsenicalism were not evident. Her haemoglobin was 76 per cent, liver function excellent, serum albumin 5.0, pseudocholinesterase 55 units and prothrombin 100 per cent. October 1952 porta-caval anastomosis of 15 mm in diameter, portal venous pressure being reduced from 295 mm to 230 mm water. Her post-operative recovery was excellent, marred only by ankle oedema and obesity. Since then serum albumin average 4.3, pseudocholinesterase 56 units, haemoglobin often exceeding 100 per cent.

Case No 102 E T FEMALE AGED 44. Moderate cirrhosis following infective hepatitis two years previously.—Five weeks before admission severe haematemesis and melaena. On examination cirrhotic liver and enlarged spleen. Haemoglobin 52 per cent, serum proteins albumin/globulin 4.5/3.9, pseudocholinesterase 30 units, serum bilirubin 1.6 mg. Her gastro-oesophageal varices are illustrated in Fig 46 p 53. November 1952 porta caval anastomosis of 19 mm diameter. Portal venous pressure reduced from 410 mm to 155 mm water. Recovery was excellent. Since then her health has been good and she has been at work as an entertainer. The serum albumin has fluctuated between 3.5 and 5.4, the pseudocholinesterase between 15 and 53 units and the bilirubin between 2.5 and 4.3 mg/100 ml.

Case No 104 E B MALE AGED 41. Arsenical cirrhosis of moderate extent.—At age of 21, twenty years before admission, suffered from *petit mal*. For thirteen years this was treated with a mixture containing arsenic and bismuth. Hyperkeratosis of the palms of the hands and soles of the feet had been noticed for five years. A wart appeared on his back one year before admission. Nine months before he had a haematemesis requiring transfusion of 4 pints of blood and two months before repeated haematemeses and melaena requiring transfusion of 7 pints. Laparotomy elsewhere had shown that the liver was nodular and cirrhotic and the spleen large. On examination there were scattered spider naevi, liver and spleen were both palpably enlarged. To the left of the midline in the lumbar region there was a rodent ulcer nearly one inch in diameter. The palms and soles showed gross hyperkeratosis with horny excrescences (Fig 29 p 35). Barium swallow showed large oesophageal varices. Liver function tests fair, albumin/globulin 3.4/3.6, pseudocholinesterase 88 units. November 1952 porta caval anastomosis. The venogram is illustrated in Fig 19 p 23 to show the earliest hypothetical stage in the development of a cavernoma. A stoma of 15 mm was constructed. The portal venous pressure dropped from 290 mm to 240 mm water. The liver showed the typical histological appearances of portal cirrhosis.

After operation his fits increased and he returned to a regime of phenobarbitone and sodium amytal and epanutin.

His physical condition during the last three years has been good. His liver function has been excellent, serum protein improving to albumin/globulin 5.7/1.2 with a pseudocholinesterase of 108 units, haemoglobin up to 120 per cent.

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In November 1956 seven malignant tumours of his skin were excised three rodent ulcers of the forehead three intra epidermal carcinomata on the right forearm left calf and left hand and one squamous-celled carcinoma of the right thigh More are forming

Case No 106 H F MALE AGED 52 Advanced cirrhosis following infective hepatitis at the age of 13—Also as a young man he had been treated with arsenical preparations for lichen planus Fifteen months before admission his health had begun to deteriorate and for five months his abdomen had been swelling enormously On examination he was grossly ascitic and showed all the stigmata of cirrhosis hepatis Liver function poor serum proteins albumin/globulin 2.8/3.8 pseudo cholinesterase 30 units There was no clinical evidence of chronic arsenicalism

He was given intensive medical treatment and for the first two and a half months he formed ascitic fluid at the rate of 15 oz a day A large right pleural effusion then suddenly appeared and all further tapplings were done of the right chest so called pleural fluid being removed at the rate of between 20 and 30 oz a day No further abdominal paracentesis was done and it was assumed that he had developed a peritoneal pleural fistula and that his ascitic fluid drained up into his right pleura After a further month of medical treatment his liver function had improved so that the serum proteins albumin/globulin were 3.4/3.3 After still further medical treatment, in April 1953 the operation of common hepatic and splenic arterial ligation was done Portal venous pressure was 230 mm water with a systolic arterial pressure of 70 mm Hg before and 260 mm water with systolic blood pressure of 130 mm Hg after the arterial ligation Post-operatively the patient held his own and it appeared possible that he might recover but on the twelfth post operative day he had a massive haemorrhage and died

Case No 108 E W FEMALE AGED 65 Advanced cirrhosis following biliary obstruction due to gallstones—Multiple haemorrhages and jaundice for two years before admission Liver function poor serum proteins albumin/globulin 3.0/4.4 pseudocholinesterase 14 units She had intensive medical treatment for one month and then in January 1953 porta caval anastomosis was done and a large solitary gallstone was removed from the common bile duct which was then closed On dissecting out the portal vein it was found to lie between the two branches of the hepatic artery The posterior artery hooked round its outer surface in a manner similar to that illustrated in Fig 4 p 6 To add to the difficulties of the dissection the portal vein was found to be partially thrombosed However a reasonable anastomosis was constructed but on releasing the clamps it was found to be kinked over the posterior hepatic artery

Post-operatively the patient did well for five days and then had a massive haematemesis She lapsed into coma and died on the seventh post operative day following further haemorrhage Postmortem it was found that the portal vein was completely occluded by thrombus between the hepatic artery and the stoma The alimentary canal was full of blood

Comment—In this case too much was attempted at one operation. In the first place no more than choledochotomy should have been done. At a second stage operation two to four weeks later a hepato renal anastomosis would have been preferable to the porta-caval.

Case No 110 I M FEMALE AGED 60 Advanced cirrhosis of unknown aetiology associated in the first place with myeloclerotic anaemia, then polycythaemia and finally myeloid leukaemia—Her complicated story began fourteen years before admission when she had her first massive haematemesis requiring transfusion. Haemorrhages continued at long intervals until two months before admission when she had a haematemesis requiring the transfusion of 12 pints of blood. On admission she was very thin and anaemic with an enormous liver and spleen. By percussion and palpation the spleen could be measured to be 14 inches in length (see Fig 22 p 28). Haemoglobin 18 per cent, serum proteins albumin/globulin 4.1/1.4, pseudocholinesterase 39 units. Medical treatment was given for seven weeks during which time 20 pints of blood were transfused and the haemoglobin was raised to 40 per cent. It was decided that splenectomy should be the first step in her treatment. This was done in February 1953. This proved to be an operation of exceptional difficulty the spleen consisting of four separate parts each firmly adherent to surrounding structures. During the operation 4½ pints of blood were transfused. The cardiac pouch of the stomach was inadvertently removed between the upper two spleens. The defect was recognised and closed. Section of the rib removed to gain access to the spleen confirmed the diagnosis of myeloclerosis and the spleen itself showed extramedullary haemopoiesis.

At splenectomy the portal venous pressure had been measured at 255 mm water and large retroperitoneal veins demonstrated (see Fig 35 p 45). She made an excellent recovery from this hectic splenectomy and five weeks later porta-caval anastomosis was done. A satisfactory stoma of 18 mm in diameter was constructed but the portal pressure remained at 270 mm water after the anastomosis.

She recovered well and returned to normal activities. Without further transfusion her haemoglobin at one time reached 114 per cent. Her serum proteins fluctuated but on an average the readings were albumin/globulin 4.5/2.5. Her pseudocholinesterase also improved remarkably the best figure being 94 units.

By 1955 however she had developed myeloid leukaemia. She became very ill and her haemoglobin fell to 26 per cent in 1956. Her white blood count at this time was 110 000 97 per cent of which was of the neutrophil type containing 8 per cent myelocytes. She was treated with transfusions and myleran and made a very good recovery. Liver biopsy showed the persistence of periportal connective tissue with considerable extramedullary myelopoiesis. She remains reasonably well.

Case No 111 H M FEMALE AGED 48 Advanced cirrhosis of unknown aetiology—For three years before admission she had had occasional haematemeses, one requiring the transfusion of 6 pints of blood and for nine months she had been ascitic. Paracenteses had been done at intervals of three weeks. She had all the

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signs of cirrhosis and the liver and spleen were both grossly enlarged and hard. Liver function was poor, serum proteins albumin/globulin 3 1/2:4, pseudocholinesterase 48 units. Large oesophageal varices were demonstrated on barium swallow. After nine months of medical treatment she was still bedridden and it was decided that the risk attached to porta caval anastomosis was justified. This operation was therefore done in March 1953, a stoma of 18 mm being constructed and the portal venous pressure being reduced from 305 mm to 165 mm water. She made an uninterrupted recovery and returned to normal existence as a housewife.

One year later she was readmitted for a right herniorrhaphy. She was then in good health with much improved liver function, serum proteins albumin/globulin 4 2/2:6. Her present health remains good, haemoglobin 105 per cent compared with 74 per cent pre-operatively, serum proteins albumin/globulin 4 3/2:7. She has however developed a low grade jaundice with a serum bilirubin of 3.5 mg/100 ml.

Case No 112 M R S MALE AGED 45 Moderately advanced cirrhosis of unknown aetiology.—Four months before admission an enlarged liver had been discovered accidentally by his firm's medical officer in South America. Three months before admission he had had a succession of haematemeses requiring blood transfusions and leading to the development of ascites. He had been invalided home. On arrival he had obvious cirrhosis and was in a poor condition. The liver was enlarged four finger breadths below the costal margin and the spleen was palpable. Free fluid was still present and a barium swallow showed large varices involving the lower half of the oesophagus. Liver function moderate as indicated by serum proteins albumin/globulin 4 0/2:3, serum bilirubin 1.5 mg/100 ml, pseudocholinesterase 39 units. Porta caval anastomosis was done in February 1953. The portal vein was found to be partially thrombosed but this did not prevent the construction of a satisfactory stoma of 15 mm in diameter. Portal venous pressure was reduced from 280 mm to 220 mm water. His post-operative recovery was excellent. He was reaccepted by his firm and returned to a full tour of duty in South America. Three and a half years later he is in very good health, liver function excellent, serum proteins albumin/globulin 5 1/2:5, pseudocholinesterase 73 units.

Case No 114 J H MALE AGED 42 Moderate cirrhosis probably resulting from an attack of infective hepatitis at the age of 15.—His cirrhosis had been diagnosed four years before admission. During the three months before admission he had had three haematemeses but his liver function remained good as shown by the serum proteins albumin/globulin 4 2:3:4. On examination he had all the signs and symptoms of cirrhosis, his spider naevi appearing like an exanthem (see Fig. 60). Barium swallow showed the presence of oesophageal varices. He had

A stoma of 18 mm in diameter was constructed and the portal

duced from 310 mm to 175 mm water. A remarkable quantity of lymph was soon seen to be oozing from the liver during the course of this operation. It clotted as it poured over the forceps during the construction of the stoma and caused considerable inconvenience.

The patient made a good recovery. His liver function three months later was satisfactory, serum proteins albumin/globulin 4.6/3.5, pseudocholinesterase 70 units. He divides his time between two farms, one in this country and one abroad, and leads a normal life.

Case No 118 F.S., MALE, AGED 37. Moderate cirrhosis attributed to severe malnutrition during three years of imprisonment in the Far East during which time he had two attacks of infective hepatitis and many other diseases, including malaria, beri beri, tropical ulcers. Two months before admission had become ascitic and icteric following four days' pyrexial illness.

He was given intensive medical treatment. During the first month his serum proteins dropped from albumin/globulin 4.0/3.9 to 3.1/2.9. After that his liver function improved until the serum proteins reached 3.8/2.6, the pseudocholinesterase was then 27 units. His condition became stationary and he ceased to form any more ascitic fluid. Barium swallow showed the presence of large oesophageal varices extending the whole length of the oesophagus. He had not bled, but it was considered that the risk of haemorrhage was such that porta caval anastomosis should be done. He was operated on in May 1953. A large stoma of 21 mm in diameter was constructed and it appeared to be functioning satisfactorily, although his portal venous pressure rose from 310 mm to 380 mm water. He made a good recovery and returned to work as a chauffeur. His liver function has improved, serum proteins albumin/globulin 4.1/1.9 being an average figure, pseudocholinesterase 46 units. His only discomfort now, three and a half years later, is a mild oedema of the ankles.

Case No 119 K.H., FEMALE, AGED 37. Extrahepatic portal vein obliteration (see Fig. 8 p. 13).—At the age of 17 she had her first haematemesis. Splenic anaemia had been diagnosed and she had been treated with a tonic containing arsenic and iron. At the age of 30 her spleen had been removed for Banti's syndrome. Haemorrhages had continued. Operation June 1953, the abdomen was explored through an oblique incision in the hope that porta caval anastomosis might be possible, but the venogram showed that there was no reasonable portal venous channel. The abdomen was closed, the patient rotated slightly to her right and an incision made along the line of the eighth left rib. Much of the oesophagus was isolated and all vascular communications to the proximal two thirds of the stomach were divided. The stomach was then transected at the cardia and re-anastomosed. The liver was noted to be mildly cirrhotic, and this was confirmed by microscopical examination.

She continued to have alimentary haemorrhage. In December 1953 a proximal gastric resection was done, the proximal two thirds of the stomach being resected.

from cardia to incisura and the oesophagus anastomosed to the pyloric end of the stomach

Since then her condition has not been satisfactory owing to regurgitant oesophagitis and reflux peristalsis. In January 1954 a temporary jejunostomy was done so that the patient's nutrition could be improved. She has had no further haemorrhage but her physical condition remains delicate. She finds eating difficult and sometimes painful but she is making steady progress.

Case No 125 S K FEMALE AGED 57 Extrahepatic obstruction due to compression of the portal vein in the porta hepatis by a primary carcinoma of the liver.—Ten months before admission became jaundiced. The liver became enlarged down to the umbilicus and the spleen almost into the left iliac fossa. She developed ascites and a caput medusae (see Fig 51 p 58). All the signs and tests indicated cirrhosis hepatis. In June 1953 the abdomen was explored with a view to doing a lienorenal anastomosis but the liver was found to be full of tumour. The spleen was therefore removed in the hope that it might relieve her of an unnecessary burden and a considerable degree of anaemia. The portal venous pressure was more than 600 mm water. A fragment of the liver taken for biopsy showed carcinoma of primary liver cell origin.

Surprisingly her jaundice disappeared and she lived for one year following this operation. Her physical condition however was very poor and she had three haematemeses before she died.

Case No 129 W G MALE AGED 39 Considered as a case of extrahepatic portal obstruction due to portal pylophlebitis (see Fig 20 p 24).—He had had infective hepatitis at the age of 22. Thirteen months before admission he had been admitted to another hospital for observation of an obscure abdominal illness. Four months later abdominal exploration showed an appendix abscess which was drained and the appendix was removed. His obscure pyrexia gradually subsided and in reviewing the case it was considered that he had had a portal pylophlebitis of septic origin. Six weeks before admission he had two severe haematemeses which required transfusion of blood. Operation July 1953 exploration with a view to doing a porta caval anastomosis (see Fig 100 p 154) but the venogram showed that the portal vein had been almost totally obliterated and recanalised. The incision was therefore extended across to the left and up through the left tenth interspace. A lienorenal anastomosis measuring 12 x 10 mm was constructed the portal venous pressure being reduced from 280 mm to 150 mm water. The speed of flow was estimated in the splenic vein 4 cm per second towards the body before anastomosis 7.5 cm per second away from the body after anastomosis.

The patient made a satisfactory recovery and has led a normal life since. His occupation is that of a plumber. Recently his liver function tests have been excellent: serum proteins albumin/globulin 4.4/2.3 pseudocholinesterase 117 units haemoglobin 114 per cent.

Case No 130 G M FEMALE AGED 52 Mild cirrhosis following infective hepatitis.—Lienorenal anastomosis June 1953 for recurrent haematemeses. She

made a good recovery but for the last two years has suffered from recurrent attacks of episodic stupor whenever she eats more than the minimum of protein. The attacks respond rapidly to treatment with glucose and sodium glutamate. Liver function shows little deterioration: serum proteins albumin/globulin 3.9/3.7, pseudocholinesterase 53 units. Between attacks she can do normal domestic work.

Case No 131 S K MALE AGED 37 Advanced cirrhosis due to inorganic arsenic—Suffered from epilepsy from the age of 21. This had been treated by numerous medicines many of which had contained arsenic. No other cause for his cirrhosis was discoverable. Six years before admission had had his first severe intestinal haemorrhage. Six months before admission had become grossly ascitic. On examination his abdomen was markedly distended with ascitic fluid, the spleen was much enlarged and the liver edge was just palpable, hard and slightly irregular. The palms and soles showed the characteristic keratosis punctata of inorganic arsenic poisoning. His complexion had been dusky for the last three years. His general condition was very poor though his liver function was no worse than fair: serum proteins albumin/globulin 3.6/2.9, pseudocholinesterase 47 units. Intensive medical treatment which had been begun elsewhere was continued but interrupted by haemorrhages. In June 1953 porta caval anastomosis was attempted in spite of portal vein thrombosis but the dissection was one of great difficulty and caused considerable haemorrhage. The operation was abandoned and 6 pints of blood were transfused. A fortnight later lieno renal anastomosis was done in spite of the fact that the splenic vein contained much blood clot which had to be scraped out before the stoma could be constructed. The patient went into hepatic coma post-operatively but recovered following the administration of streptomycin and 5 pints transfusion of blood. The ascitic fluid which had been draining from his abdomen at the rate of 2 litres a day dried up in a fortnight and he returned to heavy work as a cycle mechanic three months after operation.

For two years he remained well and at work. Liver function improving to a considerable extent as indicated by the serum proteins albumin/globulin 5.5/2.5, pseudocholinesterase 105 units. A microfilm of his chest taken when he changed his job showed that there was a shadow in the right lung. A diagnosis of bronchial carcinoma was made but the patient refused operation and died in November 1955 with massive thoracic and abdominal metastases. At postmortem the lieno renal anastomosis was found to be totally thrombosed and yet he had not developed a recurrence of his ascites. Examination of his liver and lung by activation spectroscopy showed that they contained 0.11 and 0.31 parts per million of arsenic. This is more than normal.

Case No 132 S W MALE AGED 39 Advanced cirrhosis attributed to serum hepatitis acquired during a course of antisyphilitic injections for congenital syphilitic perioritis—For seven years he had had a gradually increasing distension of the abdomen and six weeks before admission he had suddenly vomited a large quantity of bright red blood estimated at 4 pints. Two weeks before admission further haematemesis. On examination he was markedly icteric and had liver palms. Girth of 33 inches. Liver of normal size but with a hard sharp edge. Spleen

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enlarged to three finger breadths below the costal margin. Much ascitic fluid was present. A course of medical treatment was begun. Two weeks later he became ill and developed a pyrexia of 103.4° Fahrenheit. The ascites increased and he



FIG 114
Barium swallow to show the gastro-oesophageal
varices of Patient No 13

became drowsy and vomited. The next day he had a haematemesis which required blood transfusion. The comatose state responded in five days to intravenous therapy and vitamins. His abdomen required paracentesis. Soon after that he had another haematemesis and again went into coma. He became desperately ill with a very distended abdomen, deeply jaundiced and in deep coma. A barium swallow had demonstrated the extent of his oesophageal varices (see Fig 114) and his liver

according to the serum proteins appeared to be functioning surprisingly well—albumin/globulin 4.3/3.4, pseudocholinesterase 34 units

Urgent porta caval anastomosis was decided upon as the only possible means of saving this man's life. It was done five weeks after admission. The liver was very small and grossly nodular, the spleen much enlarged. More than 10 pints of ascitic fluid were withdrawn from the peritoneal cavity. An end-to-side porta-caval anastomosis of 16 mm in diameter was constructed without difficulty, the porta venous pressure being reduced from 340 mm to 160 mm water.

Post operatively the patient did well but on the sixth day he became jaundiced and drowsy. He was given 10 per cent glucose with sodium glutamate. He recovered consciousness but developed marked oedema of both legs, the scrotum, and lumbar region. From time to time he became irrational and sometimes even maniacal, but this mental state appeared to improve remarkably when glutamate was given continuously. He was discharged on the thirty-eighth post-operative day.

Three months after operation he returned to work. His jaundice had faded, there was no ascites and his general health was excellent, serum proteins albumin/globulin 4.4/2.4, pseudocholinesterase 44 units, serum bilirubin 2.8. The serum proteins improved even further, the serum albumin reaching 5.0 over a period of six months.

Twenty months after porta-caval anastomosis he had a dental extraction with considerable haemorrhage from his gums which lasted for twelve hours. His mental state became confused and he was readmitted for two weeks' treatment with glucose and glutamate intravenously, which brought about a rapid improvement in his condition and he was discharged on a low protein diet.

Three months later his jaundice recurred and he became drowsy. He was admitted in incipient coma, deeply jaundiced, with a serum bilirubin of 29 mg per cent, serum proteins albumin/globulin 3.9/3.1, pseudocholinesterase 35 units. He was given the full treatment for hepatic coma, but his state deteriorated and he died almost exactly two years after porta caval anastomosis. Postmortem his liver showed subacute hepatic necrosis and was markedly shrunken and lobular. Bronchopneumonia was present. The biliary tract was intact and normal. The porta caval anastomosis was smooth and of full size.

Case No 138 M. S., FEMALE, AGED 44. Advanced Idiopathic cirrhosis.—Twenty and ten months before admission she had had two large haematemeses, after the second of which she had become grossly ascitic. She had required frequent paracenteses. On examination she had all the stigmata of cirrhosis (see Fig. 61A, p. 76) a distended ascitic abdomen with palpably enlarged hard liver and spleen. She also had a left sided pleural effusion. Liver function tests were poor as indicated by the serum proteins albumin/globulin 2.5/4.2, pseudocholinesterase 50 units.

A course of medical treatment was begun and a month later the serum albumin had improved to 2.9 but the pseudocholinesterase had become reduced to 27 units. Soon after this her condition declined and she developed coma and died. The

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pseudocholinesterase had given a better indication of the future course of events than any other liver function test

Case No 141 V B FEMALE AGED 44 Primary biliary cirrhosis with Cruveilhier Baumgarten syndrome (see Fig 49 p 56)—Two years before admission developed severe psoriasis. For this she was treated among other things with a mixture containing liquor arsenicalis but the amount of arsenic was negligible. Eighteen months before admission she became jaundiced. Her liver and spleen both became enlarged. She was given three months' bed rest with intensive medical treatment but without improvement. On examination she was deeply jaundiced the liver was enlarged three finger breadths below the costal margin and the spleen two finger breadths. There was no ascites. Liver function moderate as indicated by serum proteins albumin/globulin 3.9/3.1 *pseudocholinesterase* 44 units serum bilirubin 4.4 mg per cent. Barium swallow failed to reveal the presence of oesophageal varices but they were seen on oesophagoscopy.

Her failure to improve under medical treatment and the absence of proof that her jaundice was not obstructive compelled abdominal exploration. This was done in November 1953. The portal venous pressure was measured at 260 mm water and an obvious cirrhosis without biliary obstruction was demonstrated. The umbilical vein measured 17 mm in diameter. It was decided to construct a porta caval anastomosis to relieve the portal hypertension and to attempt to reduce the congestion within the liver. A stoma of 16 mm in diameter was constructed and the venous pressure dropped to 150 mm water. The histological picture of the liver biopsy specimen suggested a diagnosis of post necrotic scarring. She made a good recovery and the serum bilirubin became temporarily reduced to 3.1 mg per cent.

Her progress was satisfactory with some improvement in liver function though her jaundice fluctuated. She became stronger in herself but a year after operation had a melaena stool and became comatose. She made a very rapid recovery and a barium swallow failed to reveal the presence of any varices but the meal disclosed a large duodenal ulcer.

Since then her state has remained practically unchanged and she has managed to lead a reasonably normal life.

Case No 142 S S MALE AGED 34 Idiopathic cirrhosis of moderate extent—This man was quite well until two months before admission when he had an effortless haematemesis. He was transfused 12 pints of blood at another hospital. An exploratory operation was carried out in the belief that the blood was coming from a duodenal ulcer but no ulcer was found and the liver was found to be cirrhotic. A Sengstaken tube was passed but failed to control the haemorrhage. This continued after transfer to St Bartholomew's Hospital and required the transfusion of a further 7 pints of blood. By this time he had become grossly ascitic with a girth at the umbilicus 48 inches and semi comatose. In November 1953 an emergency oesophagotomy with ligature of the bleeding oesophageal varices was done through the bed of the eighth rib. During this operation 4 pints of blood were

transfused but he was returned to the ward in excellent condition and the bleeding ceased. He was given a high protein diet through the Ryle's tube and then by mouth and rapidly built up to a reasonable state of health. serum protein albumin/globulin 3 1/3 4 pseudocholinesterase 27 units

His condition however was considered to be precarious and exactly a fortnight after the oesophagotomy porta caval anastomosis was done. A stoma of 20 mm in diameter was constructed. The portal venous pressure was reduced from 220 mm to 140 mm water and the speed of flow in the portal vein as measured by the scintillation detector and radioactive sodium increased from 4.3 cm per second to 12.5 cm per second (Fig. 79A).

He again made a good recovery from operation. Five weeks later he became drowsy but recovered with glucose and glutamate.

He returned to work as a sales representative but remained at the central office rather than travelling around the country by car.

He has frequently had attacks of episodic stupor. On one occasion for example he left the office to return to the Crystal Palace and found himself at Folkestone. He was inarticulate with panic but managed to find his way home. He has discovered that an almost totally vegetarian diet is the best and when he feels an attack of stupor about to develop he takes tetracycline 250 mg six hourly for five days. He also finds amphetamine of value to keep him awake during the day time and sometimes requires a mild sedative for sleep at night. He is however able to hold down a good job and lead a practically normal life. His ascites has not recurred since porta caval anastomosis and his abdominal girth remains at 32 inches a reduction of 16 inches. Liver function has remained stationary for the last three years. serum proteins albumin/globulin 4 1/2 8 pseudocholinesterase 51 units with minor fluctuations only.

Case No 146 A III MALE AGED 26 Idiopathic cirrhosis of mild extent— During the four years before admission had had four massive haematemeses requiring transfusion of large quantities of blood. After the second haemorrhage his abdomen had been explored elsewhere a cirrhotic liver had been discovered and the right and left gastric and gastro epiploic arteries had been tied. Porta-caval anastomosis was done in January 1954 the stoma measured 19 mm in diameter. The portal venous pressure was reduced from 390 mm to 310 mm water and the speed increased from 4 cm to 7 cm per second. He had a small haematemesis immediately after operation but made a good recovery.

Six months later he had a melaena stool but barium swallow showed that there were no oesophageal varices (see Fig. 77 p. 120). However the barium meal showed that he had a very large duodenal ulcer. This persisted until fifteen months after porta-caval anastomosis. He was treated with a milk drip down a Ryle's tube. For the last four months he has been in excellent health and has been able to return to full work.

Case No 149 P T MALE AGED 6 Severe idiopathic cirrhosis, possibly following infective hepatitis acquired from the mother in utero— At the age of 3

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weeks he had his first haematemesis and melaena and became jaundiced. At 3 years of age the diagnosis of cirrhosis hepatis with portal hypertension was established. At 4½ he developed gross ascites and the haemorrhages became more frequent. The liver was palpable three finger-breadths below the costal margin. When first seen, eight months before admission, he was desperately ill, bedridden with ascites, considerably jaundiced, and having a haematemesis with every minor ailment. For three months the serum albumin fluctuated between 3.1 and 2.3 (see progress chart, Fig 68, p 94). Finally, with intensive medical therapy, he began to improve and in June 1954 he was considered fit enough for porta caval anastomosis. A stoma of 10 mm in diameter was constructed. The portal venous pressure was reduced from 450 mm to 295 mm water and the speed of flow in the portal vein increased from 6.5 cm to 7 cm per second. The liver was remarkable in that it was a smooth, pale, tough, round organ quite unrecognisable as liver. Microscopically, however, the remaining liver cells were very healthy (Figs 23 and 24, pp 29 and 30).

The patient made a good recovery, lost his ascites, became less icteric, and was able to go to school for the first time in his life. In the beginning he required special tuition, but rapidly caught up with his elder brother both at school and physically until he outgrew him and passed him in class. His health is now excellent, serum proteins albumin/globulin 4.2/2.2, pseudocholinesterase 55 units. The serum bilirubin is still somewhat elevated at 2.8 mg per cent.

Case No 151 J. B., MALE, AGED 31 Idiopathic cirrhosis, mild.—Between 1945 and 1949 had repeated haematemeses from oesophageal varices. In 1949 lieno renal anastomosis was done by another surgeon. The liver was examined and passed as normal. No portal venogram was taken and a diagnosis of congenital extrahepatic portal vein obliteration was made on inadequate grounds. Following this operation he was well for three and a half years, when he had another massive bleed. On examination the only stigmata of cirrhosis were the presence of a few spider naevi and a faint foetor hepatis. On the strength of these findings in January 1954 the abdomen was explored for porta caval anastomosis, the diagnosis of cirrhosis was confirmed, a Crueilhier Baumgarten syndrome was disclosed (see Fig 53 p 60), and a porta-caval anastomosis was constructed. The diameter of the stoma was 17 mm. The portal venous pressure was reduced from 250 mm to 125 mm water. The speed of flow in the portal vein was increased from 4.5 to 8 cm per second. The speed of flow was also measured before the shunt in the umbilical vein and was only 1.5 cm per second. This was the first time that positive proof had been obtained of the sluggishness of the flow in the natural collateral anastomotic channels. The total operating time, including investigations, photographs etc. was only two and a half hours.

The patient made a very good recovery from this operation. Most of the spider naevi disappeared on the seventh post operative day. He returned to full work as a master builder and has remained at work since. Serum proteins however, remain at albumin/globulin 3.7/3.0, the pseudocholinesterase at 73 units.

APPENDIX 5

Case No 155 T B, MALE AGED 23 **Idiopathic cirrhosis, mild, with total thrombosis of the portal vein**—For six years before admission he had had many

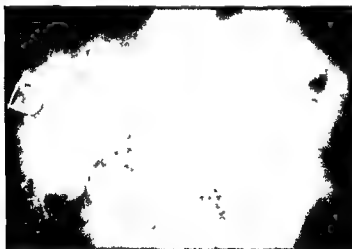


FIG 115

Patient No 155 Double venogram to show almost total thrombosis of the portal vein

severe gastro-oesophageal haemorrhages Three and a half years before he had been explored at another hospital and his spleen had been removed Fifteen months before admission he had developed acute intestinal obstruction Further

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abdominal exploration had demonstrated mesenteric thrombosis for which a large segment of jejunum was resected. He then had further gastro oesophageal haemorrhages. Palmar blush was the only stigma of cirrhosis. Liver function was good as indicated by the serum proteins albumin/globulin 5.3/2.2 pseudocholinesterase 58 units. In March 1954 the abdomen was explored with a view to doing porta caval anastomosis. The venogram however showed that the portal vein was totally thrombosed nor was there any other venous channel of any use for portal systemic venous anastomosis. It was decided that an attempt should be made to dissect the portal vein clear it of clot and construct an anastomosis but this proved to be impossible and the incision was closed. He went home and had more haemorrhages. In July 1954 he was readmitted for proximal gastric resection. This operation was done through the bed of the left eighth rib the pyloric end of the stomach being anastomosed to the distal cut end of the oesophagus. The patient made a good recovery and since gastric resection his general health has been excellent and he has been at full work. Now his haemoglobin is 100 per cent serum proteins albumin/globulin 5.3/1.8 pseudocholinesterase 61 units.

Case No 157 D D MALE AGED 24 Extrahepatic portal obliteration due to portal pylephlebitis secondary to appendix abscess—Three years before admission he had had an operation for a perforated gangrenous appendix. A swinging fever had persisted for six weeks post operatively and he was thought at one time to be suffering from a right perinephric abscess. Exploration of the right kidney did not disclose any suppuration and it was later concluded that his fever had been due to portal pyaemia. Eleven months before admission he had his first massive gastro oesophageal haemorrhage. Oesophageal varices were present on X ray. On examination he was a healthy normal young man with no evidence of liver disease. March 1954 lienorenal anastomosis. The venogram is illustrated in Fig 16 p 70 and it shows obliteration of the portal vein by old thrombus. An anastomosis of only 11 mm in diameter was constructed. The portal venous pressure was reduced from 300 mm to 210 mm water and the speed altered from 6 cm per second towards the body to 7.5 cm per second away from the body down the splenic vein. He made an uninterrupted recovery from this operation and then emigrated to Kenya. Since then he has been farming with great success.

Case No 159 W O MALE AGED 56 Following gold injections for ankylosing spondylitis, cirrhosis of moderate extent associated with a duodenal ulcer—Seven weeks before admission had a prolonged persistent haematemesis. Exploration at another hospital had revealed the presence of a moderately advanced cirrhosis together with a duodenal ulcer. In the past during the twenty six months between May 1939 and June 1941 he had had three courses of gold treatment at another hospital with the injection of a total of 3.88 gm of various gold salts which contained 1.75 gm of metallic gold. He had also had the 'Russian' treatment for ankylosing spondylitis deformans with iatrogenic serum but this had been ineffective. On examination he had the rigid stooped body of ankylosing spondylitis deformans. The liver was palpable two finger breadths below the costal margin.

its surface hard and irregular. In April 1954 porta-caval anastomosis was done. The portal venous pressure was only 120 mm water and the portal venogram demonstrated marked portal stasis (see Fig 58, p 69). The stoma measured 18 mm in diameter and the portal venous pressure after the anastomosis was 130 mm water. He made a good recovery and returned home to his usual life.

In July 1954, three months after operation, he had an attack of episodic stupor from which he made a good recovery with medical treatment. His liver function as illustrated by the serum proteins albumin/globulin 4.1/3.1 before operation, improved to albumin/globulin 4.5/2.9 six months after operation. Fourteen months after operation, however, his condition suddenly deteriorated, he became deeply jaundiced, comatose, and died. Postmortem he was discovered to have primary carcinoma of the liver (see Fig 30, p 37). The porta-caval anastomosis was in a very satisfactory state (Fig 98 p 151).

The quantity of gold present in the liver was measured by radiation spectroscopy. The cirrhotic liver contained 23.8 parts per million and the cancerous liver only 1.3 parts per million.

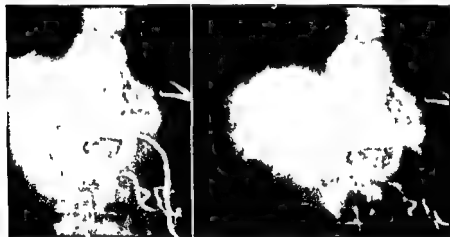


FIG. 116

Case No. 160. Double venogram through left gastro-epiploic vein in a case of mild cirrhosis showing a tortuous splenic and a large portal vein: the portal more suitable for a shunt.

Case No. 160. A H. MALE AGED 29. A case of mild idiopathic cirrhosis.—Seven years before admission had had a haematemesis associated with splenic anaemia. Deterioration of his condition led to the conclusion that operative treatment was necessary. In May 1954 he was explored with the intention of doing a lienorenal anastomosis but an excellent portal vein was disclosed on venography and a porta-caval anastomosis was constructed instead. The transverse incision is illustrated in Fig 94 p 144. The double venogram in this case is shown in Fig 116. The portal vein is obviously more suitable for anastomosis than the splenic

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Oesophageal and peri oesophageal varices are well shown. A stoma of 18 mm. in diameter was constructed. The patient made an excellent recovery. He soon returned to work and has remained fit since. His liver function has been normal throughout.

Case No 164 E G FEMALE AGED 23 Idiopathic cirrhosis of moderate extent—She had her first haematemesis and melaena at the age of 13 nearly eleven years before admission. The liver and spleen were then noticed to be markedly enlarged. She had another large haematemesis nine years before admission when her spleen extended to three finger breadths below the left costal margin and the liver to four below the right. Splenectomy was done and the liver was seen to be very cirrhotic.

She was well for two years and then had a large haematemesis. Another occurred one month before admission requiring a transfusion of 2 pints of blood and yet another ten days before admission requiring 12 pints. On examination she was noticed to have spiders of various types over the distribution of the superior vena cava particularly over the sternum as shown in Fig 61D p 77 and the white nails of cirrhosis (Fig 64 p 79). On 5th May 1954 porta-caval anastomosis was done. The size of the stoma was little more than 1 cm in diameter the portal vein itself being long and narrow as shown by the venogram (Fig 38 p 48). The pressure before the shunt was 170 mm water after 215 mm and the speed of flow 2.5 cm per second before and 5.5 cm after.

She made a good recovery from this operation and was free from haemorrhage for eleven months. She then had another severe bleed requiring transfusion. In May 1955 proximal gastric resection was done with stripping of the lower oesophagus. The portal venous pressure was 245 mm water and venography showed that the previous porta caval anastomosis had become occluded. The picture on this occasion (see again Fig 38) showed the full extent of the anastomotic channels around the diaphragm and extending up the left phrenic vein. Unfortunately she developed a subphrenic abscess post operatively which developed into a gastric fistula. A jejunostomy was done the abscess healed and the fistula closed. However the liver condition deteriorated considerably and she became ascitic. She developed intermittent pyrexia for which no cause could be found. The subphrenic space on the left side was re explored without discovering any evidence of a localised infection. She finally died in hepatic coma in December 1955.

Case No 165 R H MALE AGED 37 Idiopathic cirrhosis mild—For ten years before admission had had repeated haematemeses and melaena. Splenectomy had been done seven years before and at operation it was discovered that the splenic vein was thrombosed. Three years before admission to St Bartholomew's he had again been explored. Adhesions of the most astonishing density and vascularity were discovered and the operation had been abandoned. In June 1954 laparotomy was again done. The portal venous pressure was 205 mm water. Portal venogram showed that the portal splenic and superior mesenteric veins had been thrombosed in the past and recanalised. Further dissection proved that

there was no vein suitable for anastomosis. The speed of flow up the cavernoma was 3 cm per second. The liver showed Laennec's cirrhosis and a fragment was taken for biopsy. The operation was abandoned and it was decided that proximal gastric resection should be done in the near future. Microscopical examination of the fragment of liver confirmed the macroscopical appearance.

In July 1954 through a left thoracotomy incision the diaphragm was divided and the proximal end of the stomach approached. Anastomotic channels could be seen communicating between the portal vessels below the diaphragm and pulmonary veins above (see Fig 39 p 48). 'The proximal part of the stomach was mobilised with great difficulty owing to the vascularity and density of the adhesions. In fact so great were the hazards that it was found possible only to effect a transection and not the resection which had been hoped would be possible. The oesophagus was divided between clamps the cardia closed and the distal end of the oesophagus implanted into the fundus. The diaphragm and the pleural cavity were closed with appropriate drainage.' To give an idea of the difficulties of this operation of which the above is a direct transcription from the operation note summary it should be mentioned that it took two hours to find the stomach at all. However the patient made a good recovery and has emigrated to Australia. He has kept in touch and has had no further haemorrhage. Fig 83 p 131 shows the manner in which this transection operation has been effective in eliminating his oesophageal varicosities.

Case No 166 M M MALE AGED 49. *Idiopathic cirrhosis of moderate extent*—Six months before admission had haematemesis and melaena requiring the transfusion of 6 pints of blood. This was followed by the development of ascites and ankle oedema. There was another haemorrhage three months before admission. On examination spider naevi were present and his liver and spleen were palpable. Porta caval anastomosis was done in May 1954 the portal pressure being reduced from 320 mm to 215 mm water and the speed of flow increased from 3 cm to 7 cm per second. The stoma measured 17.5 mm in diameter. He made a good recovery from the operation.

This man's liver function had been satisfactory before porta-caval anastomosis serum proteins albumin globulin 4.8:3.0 pseudocholinesterase average of 57 units. Four months later his liver function had again improved after the usual post-operative drop serum proteins albumin globulin 4.1:3.7 pseudocholinesterase 63 units but from then on the liver function steadily declined. Two years after porta-caval anastomosis the proteins had dropped to albumin globulin 2.6:3.2 and the pseudocholinesterase to 35 units. At this time he had become intensely jaundiced with a serum bilirubin of 5.7 mg per cent and an alkaline phosphatase of 27.5 *K. A. units*. He was readmitted on account of this deterioration in health and marked peripheral oedema with some ascites. In spite of intensive medical therapy he continued to decline and died in June 1956 of total liver failure and oedema thought to be due to secondary aldosteronism. A factor which had possibly contributed to this man's hepatic deterioration was that he had continued to drink stout against instructions between his discharge following porta-caval anastomosis and his readmission two years later.

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Case No 167 T P MALE AGED 57 Idiopathic cirrhosis of moderate extent.—An enlarged liver and spleen were discovered when he was being examined on account of piles. Puncture biopsy of the liver confirmed the diagnosis of cirrhosis. Liver function tests were satisfactory as illustrated by serum proteins albumin/globulin 4.7/3.2 pseudocholinesterase 95 units. Between April 1954 and March 1956 he remained in reasonable health but then he suddenly deteriorated, became grossly ascitic and required to be admitted for intensive medical therapy. The last news of him was obtained in October 1956 when he was in deep coma. An infra red photograph of his torso to illustrate some of the clinical features of cirrhosis hepatis is shown in Fig 65 p 80.

Case No 179 J R FEMALE AGED 50 Advanced cirrhosis following infective hepatitis—This patient was said to have had an attack of infective hepatitis three and a half years before admission and during the two years before admission she had had at least seven massive haematemeses. In September 1954 she had an enormous haemorrhage which was stopped with the aid of a Sengstaken tube (see Fig 70 p 96). Two days later porta caval anastomosis was done. The portal venous pressure was reduced from 440 mm. to 310 mm. water. The right hepatic artery hooked around the outer side of the portal vein crossing the cystic duct which also passed round posterior to the portal vein. The pancreas was high, hard and excessively vascular. In spite of these obstructions and a confined space a stoma of 18×7 mm was finally constructed. The operation took four hours but the patient stood up to it well and made a good recovery though jaundice which had appeared the day before operation persisted post operatively.

Two months later the jaundice was worse, serum bilirubin being 22.6 mg per cent, serum proteins albumin/globulin 3.2/4.7. The abdomen was re-explored. Portal venogram showed that the shunt was working satisfactorily and the speed of flow in the portal vein was 8 cm per second. Cholangiogram suggested that there was an obstruction at the lower end of the common bile duct and in view of this cholecystjejunostomy in Y was done. The patient recovered, but her jaundice remained unchanged and she died a month later of liver failure. Postmortem showed that the biliary passages were fully patent. There was considerable parenchymatous hepatitis on a cirrhotic liver and chronic pancreatitis. She also had terminal bronchopneumonia.

Comment—Prospects of success in this case would have been considerably better had she been operated upon earlier in the course of her disease.

Case No 180 M E FEMALE AGED 37 Arsenical cirrhosis of moderate extent—This patient was an epileptic and had taken a mixture containing arsenic for twenty two years. Three months before admission she had had a severe haematemesis requiring transfusion. A month before admission she had had a second haemorrhage. She was marked for arsenicalism (F). Her breasts were underdeveloped and there was no ascites. Her spleen was palpable three finger breadths below the costal margin and the liver

was small and hard. Oesophageal varices were present. Her liver function was good, as indicated by the serum proteins albumin/globulin 4.3/1.7, pseudocholinesterase 102 units. In October 1954 porta-caval anastomosis was done, the portal venous pressure being reduced from 400 mm to 215 mm water. The portal venous speed increased from 5 cm to 9 cm per second. The stoma measured 17 mm in diameter.



FIG 117

Case No. 180 Rainsdrop pigmentation under-developed breasts scanty body hair of chronic arsenicalism with cirrhotic hepatitis

As a precaution against post operative infection she was given a course of aureomycin. As a result of this she developed staphylococcus and monilia enteritis and septicaemia from which she died. (This case has been reported by Matthias and Rees.)

Case No. 183 I.C., MALE, AGED 41. Portal vein thrombosis probably traumatic in origin.—For seventeen years before admission i.e. since 1937, he had had many haematemeses, at least one a year. In 1938 splenectomy had been done without affecting the course of his disease. In November 1954 the abdomen was

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Case No. 167 T P, MALE, AGED 57 Idiopathic cirrhosis of moderate extent—An enlarged liver and spleen were discovered when he was being examined on account of piles. Puncture biopsy of the liver confirmed the diagnosis of cirrhosis. Liver function tests were satisfactory, as illustrated by serum proteins albumin/globulin 4.7/3.2, pseudocholinesterase 95 units. Between April 1954 and March 1956 he remained in reasonable health, but then he suddenly deteriorated, became grossly ascitic, and required to be admitted for intensive medical therapy. The last news of him was obtained in October 1956 when he was in deep coma. An *infra red* photograph of his torso to illustrate some of the clinical features of cirrhosis hepatitis is shown in Fig 65 p 80.

Case No. 179 J R, FEMALE, AGED 50 Advanced cirrhosis following infective hepatitis.—This patient was said to have had an attack of infective hepatitis three and a half years before admission, and during the two years before admission she had had at least seven massive haematemeses. In September 1954 she had an enormous haemorrhage which was stopped with the aid of a Sengstaken tube (see Fig 70 p 96). Two days later porta caval anastomosis was done. The portal venous pressure was reduced from 440 mm to 310 mm water. The right hepatic artery hooked around the outer side of the portal vein crossing the cystic duct which also passed round posterior to the portal vein. The pancreas was high, hard, and excessively vascular. In spite of these obstructions and a confined space, a stoma of 18 × 7 mm was finally constructed. The operation took four hours but the patient stood up to it well and made a good recovery, though jaundice, which had appeared the day before operation, persisted post operatively.

Two months later the jaundice was worse, serum bilirubin being 22.6 mg per cent, serum proteins albumin/globulin 3.2/4.7. The abdomen was re-explored. Portal venogram showed that the shunt was working satisfactorily and the speed of flow in the portal vein was 8 cm per second. Cholangiogram suggested that there was an obstruction at the lower end of the common bile duct, and in view of this cholecystojejunostomy in Y was done. The patient recovered but her jaundice remained unchanged and she died a month later of liver failure. Postmortem showed that the biliary passages were fully patent. There was considerable parenchymatous hepatitis on a cirrhotic liver and chronic pancreatitis. She also had terminal bronchopneumonia.

Comment—Prospects of success in this case would have been considerably better had she been operated upon earlier in the course of her disease.

Case No. 180 M E, FEMALE, AGED 37 Arsenical cirrhosis of moderate extent.—This patient was an epileptic and had taken a mixture containing arsenic for twenty years. Three months before admission she had had a severe haematemesis requiring transfusion. A month before admission she had had a second haemorrhage. On examination she had the typical raindrop pigmentation of chronic arsenicalism (Fig 117) with liver palms and white nails. Foetor hepatis was marked. Her breasts were underdeveloped and there was little pubic hair present. Her spleen was palpable three finger-breadths below the costal margin and the liver

tion was one of some misery so it was decided that porta caval anastomosis should be done. A stoma of 18 mm was constructed the portal venous pressure reduced from 330 mm to 140 mm water and the speed of flow in the portal vein increased from 10 cm to 12 cm per second. Following this operation he made a good recovery the ascites disappeared and he was discharged on the twenty first post operative day.



FIG 118

Case No 195 Oesophageal varices still present after proximal gastric resection and stripping of the oesophagus

Fifteen months later his duodenal ulcer perforated he was operated upon and died on the second post operative day in hepatic coma. At postmortem the advanced degree of cirrhosis was demonstrated. The porta caval anastomosis was fully patent the size of a little finger and did not show any sign of thrombosis.

Case No 195 M B FEMALE AGED 34 Portal vein obliteration with cavernoma formation considered to be congenital in origin—Two and a half years before admission she had begun to have severe haematemesis. A year before gastric

PORTAL HYPERTENSION

transection had been done elsewhere without affecting the haemorrhages. In January 1955 the abdomen was re explored. A venogram showed that the portal and splenic veins had been totally obliterated and confirmed the presence of massive gastro oesophageal varices which had been seen on the barium swallow (see Fig 45 p 53). The portal venous pressure was 240 mm water. The proximal half of the stomach was resected and the oesophagus was stripped up to behind the root of the lung. It was re anastomosed to the pyloric end of the stomach. The patient made a very satisfactory recovery but one year later had further haematemesis. A barium swallow in 1956 showed that oesophageal varices are still present but that the stomach now appears to be free. Treatment by the injection of a sclerosant solution is contemplated.

Case No 200 T R MALE AGED 47: Advanced idiopathic cirrhosis.—He was admitted in January 1955. Since the previous March his abdomen and ankles had been becoming more swollen. Recently he had been tapped five times at another hospital approximately 11 pints being aspirated on each occasion. He was a sallow man with an abdomen grossly distended with ascitic fluid. The stigmata of cirrhosis were present and after paracentesis abdominis liver and spleen were both palpable. A course of intensive medical therapy was commenced. One day his ascites appeared better and it was discovered that he had developed a right pleural effusion. Tapping of the pleural effusion relieved the ascites and it was assumed that a pleuro peritoneal fistula existed on the right side. Two and a half months after the commencement of medical treatment there was little fluid present and he was discharged. His liver function had been improved from serum proteins albumin/globulin 2.8/4.0 to 3.1/3.8 pseudocholinesterase from 12 to 26 units. Since discharge his health has remained fair and he is still able to do a little work as a pianist.

Case No 201 P P MALE AGED 49: Advanced cirrhosis following infective hepatitis.—Two years before admission had had a typical attack of infective hepatitis. During the year before admission he had noticed progressive loss of weight weakness and intermittent pyrexia. On examination there was oedema of the legs and gross abdominal distension due to ascites. Paracentesis had been done repeatedly and was done four times during the next six weeks. After a fortnight of medical treatment he had a heavy melaina stool. His liver function was reasonable as illustrated by the serum proteins albumin/globulin 3.75/3.67 but he failed to respond to medical treatment. In January 1955 therefore porta caval anastomosis was done. It was an operation of considerable difficulty but finally a stoma measuring 20 mm in diameter was constructed. The portal venous pressure was dropped from 360 mm to 210 mm water and the speed increased from 2.5 cm to 9 cm per second (Fig 79a p 122). The density and cartilaginous nature of the tissues around the portal vein made it impossible to visualise the common bile duct at any time during the operation. A small biliary leak occurred which was closed with a single fine stitch. Post operatively he became jaundiced and the blood urea rose to 192 mg per cent. He died in coma. Postmortem it was found that the common duct had unfortunately been caught up in a thread ligature.

Case No 202 M L., FEMALE, AGED 72. Alcoholic cirrhosis of advanced degree.—Four years before admission had had two haematemeses. At that time her condition had been investigated and a diagnosis of cirrhosis hepatis had been established. During the year before admission she had had two severe haematemeses. The liver was found to be grossly enlarged and hard and the spleen was palpable two finger breadths below the costal margin. Liver function was reasonable, serum proteins albumin/globulin 3.7/2.1. Since the two recent haematemeses had each produced a devastating effect on the patient, it was decided that porta-caval anastomosis would have to be done in spite of the patient's age. In February 1955 the portal vein was explored, but an abnormally placed hepatic artery, illustrated in Fig. 4, p. 6, prevented the dissection of the portal vein being completed. To add to the difficulties, the vein was partially thrombosed. The operation was abandoned and the patient died of haemorrhage during the convalescent period. Had she been younger, the alternative operation of lienorenal anastomosis would have been done as soon as the abnormality had been discovered.

Case No 205 E G., FEMALE, AGED 54. Syphilitic cirrhosis of moderately advanced degree.—Six months before admission noticed enlargement of the abdomen which had been investigated at another hospital. A cyst had been felt in the pelvis (which on retrospect could only have been a distended bladder) and a careful microscopical study of the ascitic fluid was said to have shown the presence of numerous malignant cells. A diagnosis of malignant ascites had been made and she had been treated with an injection of radioactive gold into the peritoneal cavity. There was no improvement, so the pituitary was irradiated through two temporal fields. At this time the liver was palpably enlarged three finger-breadths below the costal margin with a knobbly, hard edge and the spleen was palpable two finger breadths below the left costal margin. Liver function was reasonably good as illustrated by serum proteins albumin/globulin 3.68/1.57. The Wassermann and Kahn reactions were both positive. In February 1955 the abdomen was explored in order to arrive at a positive diagnosis. There was a hepatic lobatum. The portal venous pressure was 220 mm. water. No malignant growth was found. A nodule was removed from the liver for microscopical examination. The report read as follows:—'The area of liver examined shows foci of gummatous formation some becoming confluent with central areas of necrosis surrounded by a cellular infiltration in which plasma cells are also present. There is well marked fibroblastic reaction but only moderate changes of endarteritis. Diagnosis: syphilitic gummatous of liver.'

She was given a full course of bismuth oxychloride and penicillin lasting for four weeks. During this period and the next two months paracentesis abdominis was done seven times with an average quantity of 20 pints removed on each occasion. Intensive medical therapy for cirrhosis was also being administered. Her condition improved, the proteins reaching albumin/globulin 4.1/1.6 pseudocholinesterase 51 units. The ascites persisted. In May 1955 three months after the institution of medical treatment lienorenal anastomosis was done. In February the portal venogram had shown a normal portal vein but in May partial thrombosis had occurred.

transection had been done elsewhere without affecting the haemorrhages. In January 1955 the abdomen was re-explored. A venogram showed that the portal and splenic veins had been totally obliterated, and confirmed the presence of massive gastro-oesophageal varices which had been seen on the barium swallow (see Fig 45, p 53). The portal venous pressure was 240 mm water. The proximal half of the stomach was resected and the oesophagus was stripped up to behind the root of the lung. It was re-anastomosed to the pyloric end of the stomach. The patient made a very satisfactory recovery, but one year later had further haematemesis. A barium swallow in 1956 showed that oesophageal varices are still present but that the stomach now appears to be free. Treatment by the injection of a sclerosant solution is contemplated.

Case No 200 T R, MALE AGED 47 Advanced idiopathic cirrhosis—He was admitted in January 1955. Since the previous March his abdomen and ankles had been becoming more swollen. Recently he had been tapped five times at another hospital, approximately 11 pints being aspirated on each occasion. He was a sallow man with an abdomen grossly distended with ascitic fluid. The stigmata of cirrhosis were present and after paracentesis abdominis liver and spleen were both palpable. A course of intensive medical therapy was commenced. One day his ascites appeared better, and it was discovered that he had developed a right pleural effusion. Tapping of the pleural effusion relieved the ascites and it was assumed that a pleuro peritoneal fistula existed on the right side. Two and a half months after the commencement of medical treatment there was little fluid present and he was discharged. His liver function had been improved from serum proteins albumin/globulin 2.8/4.0 to 3.1/3.8, pseudocholinesterase from 12 to 26 units. Since discharge his health has remained fair and he is still able to do a little work as a pianist.

Case No 201 P P, MALE AGED 49 Advanced cirrhosis following infective hepatitis—Two years before admission had had a typical attack of infective hepatitis. During the year before admission he had noticed progressive loss of weight, weakness, and intermittent pyrexia. On examination there was oedema of the legs and gross abdominal distension due to ascites. Paracentesis had been done repeatedly and was done four times during the next six weeks. After a fortnight of medical treatment he had a heavy melaena stool. His liver function was reasonable, as illustrated by the serum proteins albumin/globulin 3.75/3.67, but he failed to respond to medical treatment. In January 1955, therefore, porta caval anastomosis was done. It was an operation of considerable difficulty, but finally a stoma measuring 20 mm in diameter was constructed. The portal venous pressure was dropped from 360 mm to 210 mm water and the speed increased from 2.5 cm to 9 cm per second (Fig 79b p 122). The density and cartilaginous nature of the tissues around the portal vein made it impossible to visualise the common bile duct at any time during the operation. A small biliary leak occurred which was closed with a single fine stitch. Post-operatively he became jaundiced and the blood urea rose to 192 mg per cent. He died in coma. Postmortem it was found that the common duct had unfortunately been caught up in a thread ligature.

Case No 202 M L FEMALE AGED 72 Alcoholic cirrhosis of advanced degree—Four years before admission had had two haematemeses. At that time her condition had been investigated and a diagnosis of cirrhosis hepatis had been established. During the year before admission she had had two severe haematemeses. The liver was found to be grossly enlarged and hard and the spleen was palpable two finger breadths below the costal margin. Liver function was reasonable serum proteins albumin/globulin 3.7/2.8. Since the two recent haematemeses had each produced a devastating effect on the patient it was decided that portacaval anastomosis would have to be done in spite of the patient's age. In February 1955 the portal vein was explored but an abnormally placed hepatic artery illustrated in Fig 4 p 6 prevented the dissection of the portal vein being completed. To add to the difficulties the vein was partially thrombosed. The operation was abandoned and the patient died of haemorrhage during the convalescent period. Had she been younger the alternative operation of lienorenal anastomosis would have been done as soon as the abnormality had been discovered.

Case No 205 E G FEMALE AGED 54 Syphilitic cirrhosis of moderately advanced degree—Six months before admission noticed enlargement of the abdomen which had been investigated at another hospital. A cyst had been felt in the pelvis (which on retrospect could only have been a distended bladder) and a careful microscopical study of the ascitic fluid was said to have shown the presence of numerous malignant cells. A diagnosis of malignant ascites had been made and she had been treated with an injection of radioactive gold into the peritoneal cavity. There was no improvement so the pituitary was irradiated through two temporal fields. At this time the liver was palpably enlarged three finger breadths below the costal margin with a knobby hard edge and the spleen was palpable two finger breadths below the left costal margin. Liver function was reasonably good as illustrated by serum proteins albumin/globulin 3.68/1.57. The Wassermann and Kahn reactions were both positive. In February 1955 the abdomen was explored in order to arrive at a positive diagnosis. There was a hepatic lobatum. The portal venous pressure was 220 mm water. No malignant growth was found. A nodule was removed from the liver for microscopical examination. The report read as follows—The area of liver examined shows foci of gummatous formation some becoming confluent with central areas of necrosis surrounded by a cellular infiltration in which plasma cells are also present. There is well marked fibroblastic reaction but only moderate changes of endarteritis. Diagnosis syphilitic gummatous of liver.

She was given a full course of bismuth oxychloride and penicillin lasting for four weeks. During this period and the next two months paracentesis abdominis was done seven times with an average quantity of 20 pints removed on each occasion. Intensive medical therapy for cirrhosis was also being administered. Her condition improved the proteins reaching albumin/globulin 4.1/1.5 pseudocholinesterase 51 units. The ascites persisted. In May 1955 three months after the institution of medical treatment lienorenal anastomosis was done. In February the portal venogram had shown a normal portal vein but in May partial thrombosis had occurred.

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The portal venous pressure before anastomosis was 280 mm water after 180 mm. The speed of flow in the partially obliterated portal vein was 9 cm per second. The anastomosis was end to end owing to the presence of abnormal renal arteries surrounding the renal vein. The stoma measured 10 mm in diameter. The patient returned to work as a domestic in July and in October a vaginal haemorrhage occurred. Biopsy showed that she had a squamous celled carcinoma of the cervix and a standard Wertheim's hysterectomy was done. She returned to work as a domestic again at the end of December and has remained at work since.

In July 1956 the liver function had improved the serum proteins being albumin/globulin 5.4/2.1 pseudocholinesterase 85 units.

Illustrating this case are Fig 31 p 38 32 p 39 and Fig 80A p 123.

Case No 213 L N FEMALE AGED 53 Idiopathic cirrhosis of moderate severity—Four months before admission she had had a haematemesis of approximately 1 pint of blood. Two more followed in quick succession. The abdomen was explored at another hospital a cirrhotic liver was discovered and a partial gastrectomy was done. She made an uneventful recovery. Oesophageal varices were demonstrated on barium swallow. On examination she was a thin woman with a sallow complexion and foetor hepatis. The liver was slightly enlarged and hard the spleen enlarged to three finger breadths below the costal margin. The liver function was reasonably good serum proteins albumin/globulin 4.2/2.1 pseudocholinesterase 62 units. In June 1955 porta caval anastomosis was done. The stoma measured 12 mm in diameter. The speed of flow in the portal vein was increased from 8 cm to 15 cm per second but the portal pressure which was only 160 mm water before anastomosis was 175 mm after. Splenic and portal venograms were done in this case and are illustrated in Fig 37 p 47. They show that much splenic blood was diverted via gastric varices into perirenal and pancreatic veins to the vena cava and that these same varices communicated through a large left gastric vein with the portal vein itself.

She made a good recovery from this operation but two months later had her first attack of episodic stupor. These attacks continued in spite of scrupulous attention to detail as to treatment with restricted proteins etc. The liver function continued to improve and by November 1956 the serum proteins had reached albumin/globulin 5.1/1.9 and the pseudocholinesterase 67 units having at one time been as high as 89. (She died of a cerebral haemorrhage in January 1957).

Case No 219 L C FEMALE AGED 11 Congenital portal vein obliteration with cavernoma formation (illustrated in Fig 6 p 11)—She had been having repeated haematemeses from the age of 4. Her spleen was enlarged to approximately four finger breadths below the costal margin. In July 1955 renal anastomosis was done. The splenic vein was small the renal vein was duplicated its anterior branch was clamped for some minutes without any appreciable effect on the appearance of the kidney. It was therefore divided at the renal end and a single Blalock clamp applied to the vena caval end of this anterior branch of the renal vein. It measured the same as the splenic vein 7 mm in diameter and an end to end

junction was effected between these two veins without disturbing the kidney from its bed. The portal pressure was 310 mm water before anastomosis, 230 mm after. The speed of flow towards the body was 11 cm per second before, and away from the body 7 cm per second after. She made an uninterrupted recovery and has been free from haemorrhage since.

Case No 223 F G, MALE, AGED 41. Moderately advanced cirrhosis following three years as prisoner-of-war in the Far East during which he suffered from frequent attacks of malaria, two attacks of infective hepatitis, dysentery, beri beri, starvation oedema, tropical ulcers, etc. He had had a very severe haematemesis four months before admission. He was irritable, depressed, retiring and anxious. The liver was just palpable and the spleen was enlarged to three finger-breadths below the costal margin. Spider naevi were present (see Fig 61b, p 76). Barium swallow revealed the presence of oesophageal varices. The liver function was satisfactory, serum proteins albumin/globulin 4/1.2.6. August 1955 porta-caval anastomosis. Size of stoma 21 mm in diameter. Portal venous pressure 385 mm water before, 165 mm after. Rate of flow 5 cm per second before, 7.5 cm after.

He developed diabetes post-operatively, his blood sugar rising to 334 mg per cent. This was very difficult to control and he was liable to suffer attacks of coma which were either hyper or hypoglycaemic, possibly aggravated by hepatic stupor. In 1957 he died in coma. Haemochromatosis was discovered postmortem. The serum iron and skin biopsy done during life had been negative.

Case No 229 C M, FEMALE, AGED 38. Cruveilhier-Baumgarten disease with intrahepatic portal vein obliteration (Fig 50 || 57)—Thirteen years before admission at the age of 25 she had had a massive haematemesis requiring the transfusion of 23 pints of blood and 2 pints of serum. An emergency splenectomy had been done. She had another large haematemesis fourteen months before admission when 7 pints of blood were transfused. She was fully investigated but it was decided that the patient would need to be convinced by a further haemorrhage that operative treatment was necessary. In due course the bleed occurred requiring the transfusion of 2 pints of blood with a further haemorrhage continuing for six days requiring the transfusion of 11 pints of blood. In March 1956 the abdomen was explored and the condition of Cruveilhier-Baumgarten disease disclosed with a partially thrombosed portal vein. The portal venous pressure was no more than 140 mm water. The portal vein was dissected out, divided, clot was removed from its interior, and a porta caval anastomosis of 17 mm in diameter was constructed. The pressure rose slightly to 145 mm water. Speed of flow in the portal vein was 10 cm per second before and 10 cm per second after anastomosis but it must be remembered that a fair quantity of clot was scraped out of the vein enlarging its lumen. The speed of flow in the umbilical vein was no more than 2.5 cm per second. She made a good recovery and has had no further bleeds. Her physical condition is very satisfactory.

Case No 231 R B, MALE, AGED 32. Idiopathic cirrhosis of moderate severity.—He was admitted towards the end of 1955. Six and a half years before this in

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1949 he had had two haemorrhages succeeded by a porta caval anastomosis at another hospital. He had remained fairly well until early 1955 when he began to have episodes of stupor. These were often related to severe headaches and he was prevented from doing his usual work which carried considerable responsibility. His liver function remained good except following a brief febrile illness associated with jaundice in December 1955 extending into January 1956 when the serum proteins fell to albumin/globulin 3.8/3.2. The serum proteins recovered to 4.4/2.1 pseudocholinesterase 57 units. It is difficult to tell how much of this man's trouble is due to his anxiety about himself.

Case No 232 H P MALE AGED 41 *Advanced cirrhosis resulting from starvation as a prisoner of war in the Far East*—In June 1955 this man had a haematemesis and melaena lasting seven days. After this he became ascitic. Abdominal exploration was done elsewhere and the cirrhotic liver discovered. After this paracentesis was required on many occasions, the greatest quantity of fluid removed at any one time being 15 pints. The liver function deteriorated in spite of intensive medical therapy and in the latter part of 1955 his serum proteins reached the low level of albumin/globulin 2.2/4.0. At the end of the year he had another small haemorrhage which put him into coma for nearly a week and he was thought to be dying. However he recovered continued with his medical treatment and in April 1956 his liver function had sufficiently recovered for porta caval anastomosis to be considered. This operation was done and he made an uninterrupted recovery. The serum proteins improved to albumin/globulin 4.2/3.2 pseudocholinesterase increasing from 34 to 49 units. Subsequently this improved to 66 units and he was considered fit for moderately heavy work.

Comment—The course of pre operative treatment lasted ten months.

Case No 235 S P MALE AGED 46 *Cirrhosis of moderate extent probably following an attack of infective hepatitis*—Three years before admission the liver had been seen to be cirrhotic during the course of an operation for acute appendicitis. He then developed a duodenal ulcer and the year before admission had had a haematemesis. Barium meal showed oesophageal varices. Many of the stigmata of cirrhosis were present. He had spider naevi foetor hepatis palmar blush and pale nails. There was some ankle oedema. The splenic venogram did not demonstrate the portal vein satisfactorily but the portal venogram showed it to be a good channel of full size (see Fig 66 p 85). In December 1955 a porta caval anastomosis 16 mm in diameter was constructed. The portal pressure dropped from 110 mm to 75 mm water and the speed of flow increased from 5 to 10 cm per second. A gallbladder full of gallstones also shown in Fig 66 was removed. Section of the liver fragment showed portal cirrhosis with many proliferating bile ducts. Physically he has made a good recovery from the operation but his mental state has remained sluggish. He has not done any work for four years.

Case No 239 J T FEMALE AGED 32 *Mild cirrhosis of unknown aetiology*—For five years before admission she had been anaemic. Three years before a

diagnosis of Banti's syndrome had been made and oesophageal varices were demonstrated For the year before admission she had felt tired and unwell and six days before she had had a haematemesis For the past seven years she had also suffered from piles After admission she had several haematemeses and frequent melaena stools requiring the transfusion of 10 pints of blood Her haemoglobin was 36 per cent She was given more blood transfusions On examination she was obviously ill and very anaemic with marked foetor hepaticus The spleen was enlarged four finger breadths below the left costal margin Liver function tests were excellent as illustrated by the serum proteins albumin/globulin 5.2/2.2 and pseudocholinesterase of 88 units Porta caval anastomosis was done in January 1956 without waiting for the full preparation of the patient owing to the imminent risk of continued profuse haemorrhage The percutaneous splenic venogram demonstrated a good portal vein and considerable distortion of the liver pattern proving the liver to be cirrhotic Greater backflow up gastric and oesophageal varices was demonstrated on portal venography (see Fig 47 p 54) The porta caval anastomosis measured 16 mm in diameter The portal pressure rose from 195 mm to 200 mm water and the speed of flow increased from 8 to 10 cm per second

The patient became icteric post operatively but this slowly cleared over the months Two and a half weeks after operation she developed incipient coma which responded immediately to a high carbohydrate low protein diet with sodium glutamate and intravenous vitamins Three months after operation she had almost returned to normal health and has continued to improve slowly since in spite of the presence of a mild jaundice the serum bilirubin being between 3 mg and 4.4 mg per cent

Case No 240 H H MALE AGED 44 A diabetic with advanced alcoholic cirrhosis—He had a haematemesis in June 1955 and another in November of that year He was found to be not only cirrhotic but diabetic and obese his weight being seventeen stone He had another haematemesis in December and managed to reduce his weight to fifteen and a half stone by intensive dietary treatment In 1941 he had had a thyroidectomy for Graves disease Medical treatment in this man's case was prolonged because he was considered to be a very bad risk An encouraging feature of his case was that his liver function improved and in July 1956 his serum proteins were albumin/globulin 5.7/1.9 and the pseudocholinesterase 82 units Porta caval anastomosis was done in this month the stoma measuring 18 mm in diameter but the pressure being reduced only from 220 mm to 210 mm water The liver was markedly cirrhotic and the spleen much enlarged The operation was made difficult by the enormous size of the man and the great depth of the wound

He made a good recovery and was attending to business by proxy on the tenth post operative day His liver function has remained good serum proteins albumin/globulin 4.8/3.1 and pseudocholinesterase 64 units

The venogram of this patient is illustrated in Fig 42 p 51 It shows the markedly cirrhotic state of his liver and large oesophageal and peri oesophageal

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1949 he had had two haemorrhages succeeded by a porta caval anastomosis at another hospital. He had remained fairly well until early 1955 when he began to have episodes of stupor. These were often related to severe headaches and he was prevented from doing his usual work which carried considerable responsibility. His liver function remained good except following a brief febrile illness associated with jaundice in December 1955 extending into January 1956 when the serum proteins fell to albumin/globulin 3.8/3.2. The serum proteins recovered to 4.4/2.1 pseudocholinesterase 57 units. It is difficult to tell how much of this man's trouble is due to his anxiety about himself.

Case No 232 H P MALE AGED 41 Advanced cirrhosis resulting from starvation as a prisoner-of-war in the Far East—In June 1955 this man had a haematemesis and melaena lasting seven days. After this he became ascitic. Abdominal exploration was done elsewhere and the cirrhotic liver discovered. After this paracentesis was required on many occasions the greatest quantity of fluid removed at any one time being 15 pints. The liver function deteriorated in spite of intensive medical therapy and in the latter part of 1955 his serum proteins reached the low level of albumin/globulin 2.2/4.0. At the end of the year he had another small haemorrhage which put him into coma for nearly a week and he was thought to be dying. However he recovered continued with his medical treatment and in April 1956 his liver function had sufficiently recovered for porta caval anastomosis to be considered. This operation was done and he made an uninterrupted recovery. The serum proteins improved to albumin/globulin 4.2/3.2 pseudocholinesterase increasing from 34 to 49 units. Subsequently this improved to 66 units and he was considered fit for moderately heavy work.

Comment—The course of pre operative treatment lasted ten months.

Case No 235 S P MALE AGED 46 Cirrhosis of moderate extent probably following an attack of infective hepatitis—Three years before admission the liver had been seen to be cirrhotic during the course of an operation for acute appendicitis. He then developed a duodenal ulcer and the year before admission had had a haematemesis. Barium meal showed oesophageal varices. Many of the stigmata of cirrhosis were present. He had spider naevi foetor hepatis palmar blush and pale nails. There was some ankle oedema. The splenic venogram did not demonstrate the portal vein satisfactorily but the portal venogram showed it to be a good channel of full size (see Fig 66 p 85). In December 1955 a porta caval anastomosis 16 mm in diameter was constructed. The portal pressure dropped from 210 mm to 75 mm water and the speed of flow increased from 5 to 10 cm per second. A gallbladder full of gallstones also shown in Fig 116 was removed. Section of the liver fragment showed portal cirrhosis with many proliferating bile ducts. Physically he has made a good recovery from the operation but his mental state has remained sluggish. He has not done any work for four years.

Case No 239 J T FEMALE AGED 32 Mild cirrhosis of unknown aetiology—For five years before admission she had been anaemic. Three years before a

diagnosis of Banti's syndrome had been made and oesophageal varices were demonstrated. For the year before admission she had felt tired and unwell, and six days before she had had a haematemesis. For the past seven years she had also suffered from piles. After admission she had several haematemeses and frequent melaena stools requiring the transfusion of 10 pints of blood. Her haemoglobin was 36 per cent. She was given more blood transfusions. On examination she was obviously ill and very anaemic with marked foetor hepaticus. The spleen was enlarged four finger breadths below the left costal margin. Liver function tests were excellent, as illustrated by the serum proteins albumin/globulin 5.2/2.2 and pseudocholinesterase of 88 units. Porta-caval anastomosis was done in January 1956 without waiting for the full preparation of the patient owing to the imminent risk of continued profuse haemorrhage. The percutaneous splenic venogram demonstrated a good portal vein and considerable distortion of the liver pattern, proving the liver to be cirrhotic. Greater backflow up gastric and oesophageal varices was demonstrated on portal venography (see Fig 47, p 54). The porta-caval anastomosis measured 16 mm in diameter. The portal pressure rose from 195 mm to 200 mm water and the speed of flow increased from 8 to 10 cm per second.

The patient became icteric post operatively, but this slowly cleared over the months. Two and a half weeks after operation she developed incipient coma, which responded immediately to a high carbohydrate, low protein diet with sodium glutamate and intravenous vitamins. Three months after operation she had almost returned to normal health and has continued to improve slowly since in spite of the presence of a mild jaundice, the serum bilirubin being between 3 mg and 4.4 mg per cent.

Case No 240 H. H., MALE AGED 44 A diabetic with advanced alcoholic cirrhosis.—He had a haematemesis in June 1955 and another in November of that year. He was found to be not only cirrhotic but diabetic and obese, his weight being seventeen stone. He had another haematemesis in December and managed to reduce his weight to fifteen and a half stone by intensive dietary treatment. In 1941 he had had a thyroidectomy for Graves' disease. Medical treatment in this man's case was prolonged because he was considered to be a very bad risk. An encouraging feature of his case was that his liver function improved and in July 1956 his serum proteins were albumin/globulin 5.7/1.9 and the pseudocholinesterase 82 units. Porta-caval anastomosis was done in this month, the stoma measuring 18 mm in diameter but the pressure being reduced only from 220 mm to 210 mm water. The liver was markedly cirrhotic and the spleen much enlarged. The operation was made difficult by the enormous size of the man and the great depth of the wound.

He made a good recovery and was attending to business by proxy on the tenth post operative day. His liver function has remained good, serum proteins albumin/globulin 4.8/3.1 and pseudocholinesterase 64 units.

The venogram of this patient is illustrated in Fig 42, p 51. It shows the markedly cirrhotic state of his liver and large oesophageal and peri oesophageal

varices. It was this venogram which confirmed the impression that porta caval anastomosis was essential in this case.

Case No 241 J D MALE AGED 27 Advanced cirrhosis following infective hepatitis—In 1944 at the age of 15 he had had a serious illness. He was jaundiced and ascitic and the liver and spleen were found to be enlarged. He also had acute nephritis from which he made a good recovery. In 1953 three years before admission he had had a febrile illness associated with ascites and oedema. A barium swallow at that time showed the presence of oesophageal varices. He commenced treatment with a high protein diet. Anaemia in 1954 necessitated transfusion of blood. In December of that year splenectomy was done at another hospital. In December 1955 he had had a severe haematemesis requiring the transfusion of 4 pints of blood. He again became ascitic. On examination he showed many of the stigmata of cirrhosis with spider naevi on the upper part of the chest moderate gynaecomastia and foetor hepatis. The liver was only just palpable but was very hard. He was put on a high protein diet and found he could take 170 gm a day with ease. Liver function had been poor on admission as illustrated by serum proteins albumin/globulin 3.2/3.7 but these improved steadily and rose to 3.9/3.5 pseudocholinesterase was then 34 units. In February 1956 he had a haematemesis of half a pint followed by melaena stool. He was immediately transfused and suffered little setback. In March porta caval anastomosis was done (Fig 93 p 144 illustrates the incision). The liver was very small and grossly cirrhotic. The venogram of necessity done by direct injection after the abdomen had been opened was interesting in that it showed the splenic vein was still present and communicated with many perirenal vessels (Fig 119). The portal vein was only 15 mm in diameter but by cutting it slightly obliquely it was possible to make a porta caval anastomosis 19 mm in diameter. The portal venous pressure dropped from 210 mm to 185 mm water.

On the sixth post operative day he became drowsy and could not concentrate. The protein in his diet was restricted to 60 gm a day and his mind cleared in five days. He was discharged on a diet of 80 gm protein per day without added salt.

Three months after porta caval anastomosis he was back at work as a dispenser. By August he was able to resume his previous work as the travelling representative of a manufacturing chemist.

Case No 242 D L MALE AGED 24 Congenital intrahepatic portal obstruction with Cruveilhier Baumgarten disease and thrombosis of the portal vein (see Fig 13 p 17)—This man had his first haematemesis at the age of 7 when he vomited two basinful of blood. Haemorrhages had recurred at long intervals since then the last occurring in April 1956 and requiring the transfusion of 10 pints of blood. After admission in May bleeding continued and a further 17 pints were transfused before his haemoglobin could be built up to 80 per cent. In June 1956 ilio renal anastomosis was done the venogram having demonstrated the site of the obstruction. It was felt that the umbilical vein should if possible be preserved.



FIG. 119

Double portal venogram of Case No. 741 to show backflow along splenic vein to perirenal veins of Retzius and along left gastric vein to gastric and oesophageal varices. The flow up the oesophagus is sluggish as shown by the second picture. The very small size of the liver is also demonstrated.

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to act as an additional channel for decompression. The anastomosis appeared to be very satisfactory and measured 20×16 mm. The renal vein had been so large that it had been possible to clamp off a segment of the vein and make a longitudinal incision for the stoma. The portal venous pressure dropped from 225 mm to 155 mm water and the speed of flow in the splenic vein from being 5 cm per second towards the body before construction of the anastomosis was 7.5 cm per second away from the body afterwards. The speed of flow in the portal vein was 5 cm per second and in the umbilical 2.5 cm per second.

His post operative course was complicated by an unexplained pyrexia and then much further bleeding. The chart of this man's 'post splenectomy fever' is illustrated in Fig 75 p 113. In July the condition had become desperate and exactly five weeks after the ileo renal anastomosis the abdomen was re explored. It was found that not only had the previous anastomosis thrombosed but the whole portal tree had been obliterated by thrombus. Portal venous pressure was only 125 mm water. The portal vein was dissected out and divided near the liver. No blood flowed from either end. Clot was scraped and cut out of the vein until there was a copious flow of blood. The vein was then clamped and a porta caval anastomosis measuring 23 mm in diameter was constructed.

He received a short course of heparin after this second operation. He made a good recovery and four months after porta caval anastomosis was leading a normal life doing full work with a haemoglobin of 90 per cent.

Case No 245 W P MALE AGED 36: Advanced cirrhosis due to malnutrition when a prisoner-of-war in Siam between February 1942 and August 1945.—In addition to the starvation oedema he had had pellagra beri beri tropical ulceration etc. His condition on release is illustrated in Fig 26 p 31. A year after returning to this country cirrhosis was diagnosed when he was being investigated and treated for hookworm. In December 1955 his abdomen swelled and he became grossly ascitic. Full medical treatment was instituted. He then had a severe haematemesis and his girth increased to 39 inches. His liver function deteriorated as illustrated by serum proteins albumin/globulin 2.3/4. During the next four months they gradually improved until in July they reached albumin/globulin 4.4/2.5. The pseudocholinesterase had increased from 33 to 44 units. His condition was not good but did not seem to be improving further. His girth was then no more than 34 inches. It was decided that the risk of porta caval anastomosis was less than the risk of leaving him alone. The operation was done in September 1956. Ten pints of ascitic fluid were removed. A stoma of 18 mm in diameter was constructed. The portal venous pressure was reduced from over 600 mm water to 220 mm. Venogram showed that there was no collateral circulation all the portal blood passing through the liver in keeping with the very high venous pressure. Four days after operation he became comatose and died two days later in deep hepatic coma. In the terminal phase he showed a severe potassium deficiency which was not correctable.

Case No 246 D P FEMALE AGED 46 Primary biliary cirrhosis of advanced degree—In February 1954 this patient had vomited blood and a barium meal had

demonstrated a gastric ulcer. During 1955 she had had repeated haematemesis and melaena stools and in October of that year an emergency gastrectomy had been done though no ulcer had been found at operation. After this operation she became ascitic and jaundiced, requiring paracentesis at frequent intervals. Her girth fluctuated between 34 and 36 inches. She was treated medically and made a fair recovery. Her serum proteins at this time, albumin/globulin 3/1.2, suggested that the risks of operation were too great. Bromsulphthalein retention of 36 per cent in 45 minutes appeared to confirm this suspicion. In June 1956 she had another severe haematemesis requiring many transfusions. At this time the spleen was enlarged five finger-breadths below the costal margin, the liver three. Serum bilirubin was 10.3 mg per cent. During July she had four further haematemesis requiring transfusion. By September her liver function had improved to the extent that her serum proteins were albumin/globulin 3.3/3.35. She was still grossly ascitic (see Fig. 28, p. 34). Her condition was judged to be hopeless without operation, so early in September the opportunity was taken and porta caval anastomosis was done. The portal venous pressure was reduced from 300 mm to 190 mm water. A cholangiogram demonstrated that there was no obstruction to the biliary tract. She made a very satisfactory recovery. The ascites did not recur, and after the usual temporary post-operative setback the liver function has further improved, proteins reaching albumin/globulin 3.4/2.9, serum bilirubin dropping from 14.8 to 9.2 mg per cent. Her girth is now 28 inches and she has returned to work as a teacher, having been bedridden for nearly a year before operation.

Case No. 247 H. M., MALE AGED 35. Advanced cirrhosis of unknown aetiology associated with a severe degree of nephritis.—Had a severe haematemesis four years before admission. Two days before admission began his second haemorrhage which continued for nearly a month in spite of compression with a Sengstaken tube. During this time he was transfused with more than 28 pints of blood. Oesophageal varices had been demonstrated on a barium swallow done as an emergency in bed. In April 1956 transhihoral oesophagotomy with ligation of oesophageal varices was done. The bleeding was stopped, but his ascites continued. Five weeks later he had another haematemesis lapsed into coma and died. Postmortem the advanced degree of the cirrhosis was confirmed, and an additional finding of interest was that amyloid infiltration was discovered in the liver, spleen, kidney and suprarenal glands.

Fig. 43 p. 52 illustrates the peri oesophageal varicosities in the mediastinum of this patient. Fig. 105, p. 159 illustrates the incision for oesophagotomy. The pictures illustrating this operation Fig. 109 p. 164 are tracings of photographs taken during the course of his operation. This patient shows that ligation of oesophageal varices produces no more than a temporary suppression of haemorrhage.

Case No. 248 M. H., FEMALE AGED 40. Extrahepatic obstruction due to thrombosis of the portal vein, probably infective in origin.—This patient was treated in 1956. Twenty years previously she had had a febrile illness associated with ascites which had cleared up with conservative treatment. Its aetiology had never

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been fully explained. In 1938 she had a haematemesis of 2 pints. She was then treated for splenic anaemia for several years. In December 1955 five months before admission to St Bartholomew's Hospital she had another severe haematemesis following a bout of sickness which had lasted several days. The bleeding continued for a week and required the transfusion of 10 pints of blood. Liver and spleen were found to be enlarged and the barium meal showed oesophageal varices. During March and April 1956 she had had almost continuous haematemesis and melaena. Transfusions kept pace with the blood loss but the haemoglobin could not be elevated above about 50 per cent. In May 1956 she was explored with the intention of doing a porta caval anastomosis. The condition found is illustrated in Fig 18 p 22. The portal vein had been replaced by an enormous cavernoma measuring $3 \times 2 \times 2$ inches and situated in the anterior part of the gastrohepatic omentum. The gallbladder and the common bile duct lay to the right of it and behind it. The vascular channels were so large that it was hoped that one of them might be useful for a shunt. The vena cava was dissected but the foramen of Winslow had been obliterated by adhesions and the duodenum was drawn up to the liver. On opening a space to approach the cavernoma a stony hard white rod the size and shape of an index finger was found in the place where the portal vein should have been. Everything was matted around it and it intervened between the cavernomatous channel and the inferior vena cava. This structure was presumed to be the thrombosed portal vein. The liver itself was not cirrhotic the spleen was greatly enlarged and fibrotic with many perisplenic adhesions. A splenic venogram demonstrated that a small splenic vein was present so further attempts at curative operation were deferred and later in the month splenectomy and end to side ilio renal anastomosis was done. A stoma 15 mm in diameter was constructed and the portal venous pressure was dropped from 300 mm to 130 mm water. Her recovery was uneventful. Her haemoglobin rose from 48 to 104 per cent her white blood cells from 2,000 to over 7,000 and her platelets from 60,000 to 290,000. She has been well and at work except for one bad attack of constipation which almost amounted to subacute intestinal obstruction. However it was relieved with an enema.

This patient is classified as a case of portal vein thrombosis due to an abdominal infection of obscure type. The rest of the abdominal cavity was examined particularly at the first operation and the pelvis was found to be completely obliterated with adhesions. The appendix was not involved and it was removed in passing. It showed no abnormal pathological changes.

Case No 249 J P FEMALE AGED 49 A case of mild Laennec's cirrhosis of unknown aetiology and presenting as a case of splenic anaemia—Her enlarged spleen and anaemia were discovered in 1954. A full investigation did not reveal anything else of significance. The stigmata of cirrhosis were not present and all her liver function tests were normal. She felt ill and weak and her haemoglobin sometimes dropped as low as 62 per cent. On examination the spleen was enlarged to well below the umbilicus the liver was also palpable but did not appear abnormal. Serum proteins albumin/globulin 51/23 pseudocholinesterase 137 units

thymol turbidity 5 units. Barium swallow and meal did not disclose any abnormality except for the displacement of the stomach by the enlarged spleen. She had moderate leukopenia of 3300 white cells and a platelet count of 90000. At exploration for splenectomy her portal venous pressure was found to be 370 mm water and the portal speed 4 cm per second. The liver was diffusely cirrhotic and finely granular. Venogram demonstrated diversion of portal blood into the retroperitoneal veins and down into the lumbar veins as well as back into the perinephric venous plexus (see Fig 36 p 46). Splenectomy was therefore done with ligation of all vessels as close to the spleen as possible so as to spare all these communicating anastomotic channels. The venous pressure was then found to have dropped to 195 mm water and the flow of blood in the portal vein was no longer detectable. It had not been possible by splenic venography to demonstrate the diversion of blood up the oesophagus. The patient's physical condition has been much improved by splenectomy.

Case No 250 E P FEMALE AGED 59 **Case of mild cirrhosis of unknown aetiology**—In February 1956 had a melaena stool and haematemesis which continued off and on for five days. There was nothing in the history or examination to suggest cirrhosis except for one spider naevus on the chest. She had aortic stenosis which did not prevent her from working in a fried fish shop. blood pressure 170/80. The spleen was palpably enlarged and oesophageal varices were demonstrated on barium swallow (see Fig 77 p 170). Porta caval anastomosis done in May 1956. Splenic and portal venograms showed that the splenic and portal veins were both normal. An anastomosis of 19 mm in diameter was constructed the portal venous pressure being reduced from 280 mm to 170 mm water. Speed of flow in the portal vein was 4 cm per second before anastomosis but a failure of the apparatus prevented its estimation afterwards. The liver showed a fine cirrhotic change of the Laennec type confirmed microscopically.

She made a good recovery and returned to normal life. She is now emphatic in her opinion that her health is better than it has been for five years. Her haemoglobin at 94 per cent is 14 per cent better than it was before operation. All other tests are normal except that she has a bilirubin of 1.5 mg per cent.

The post operative barium swallow shows that the oesophageal varices have become markedly less in size.

Case No 251 D W MALE AGED 53 **Extrahepatic obstruction due to invasion of the portal and splenic vein by carcinoma of the body and tail of the pancreas** (see Fig 11 p 15)—From 1953 this man had been suffering from epigastric pain after meals which was relieved by alkalis. Barium meal had revealed a duodenal ulcer and a diverticulum of the fourth part of the duodenum. The pain persisted and became more continuous and rather more in the back. He was explored elsewhere seven months before admission when the cavernoma illustrated in Fig 11 was seen. No other gross abnormality could be detected. He was then followed up closely. The pain continued he became severely anaemic and occult

blood was found frequently in the stools. Liver function was normal but the stools contained undigested muscle fibres and many fat globules the total fat being 30 per cent of the dried stool. His spleen was palpable. He was re-explored in October 1956. Splenic and portal venography showed that the splenic and distal end of the portal vein was obliterated and palpation of the pancreas showed that it contained carcinoma which was infiltrating into the surrounding tissues. There were metastases present in the left lobe of the liver and in the peripancreatic lymph nodes. Severe bleeding into the alimentary canal persisted after the operation.

Case No 258 T McQ MALE AGED 6 Congenital portal vein obliteration—Four months before admission he had a severe haematemesis. The spleen was found to descend to the level of the umbilicus and a barium swallow showed oesophageal varices. In September 1956 he was explored with a view to doing a lienorenal anastomosis. On splenic venography the diodone remained under the capsule of the spleen and failed to pass into the portal tree. Venography by way of a jejunal vein showed that the whole portal tree was obliterated and replaced by a large cavernoma. An attempt was made to dissect up some form of splenic channel following removal of the spleen but none was found. The portal venous pressure was 325 mm water before and 450 mm after splenectomy but the readings may have been falsified by a rise of arterial pressure of 30 mm Hg. He made an uninterrupted recovery and so far has had no further bleed.

Case No 261 P L FEMALE AGED 32 Mild cirrhosis associated with amyloid disease—The history in this case was complicated. In 1943 thirteen years before admission she had a feverish illness which may have been infective hepatitis. Two years later in 1945 she developed bilateral psoas abscesses with Pott's disease of the spine which required sanatorium treatment for three years. The abscesses were secondarily infected and have left very large and ugly scars in both groins. In 1950 she was treated for amyloidosis. At that time she had an enlarged liver and spleen but no proof of amyloid was obtained. In 1954 she developed tuberculous abscesses in both breasts. Two months before admission she had a severe haematemesis which required the transfusion of a large quantity of blood. This was followed by the development of ascites. She was explored elsewhere. An enlarged spleen was discovered and the diagnosis of haemorrhage from oesophageal varices was established. On examination she did not show any of the stigmata of cirrhosis: the liver was only just palpable and the spleen was enlarged four finger breadths below the left costal margin. In September 1956 porta caval anastomosis was done. Her portal pressure was very high more than 600 mm water before the shunt and 260 mm after. Splenic venogram had demonstrated patent splenic and portal veins. The liver was mildly cirrhotic with a small fibrotic plaque on its surface. The stoma measured 20 mm in diameter. Microscopical examination of the liver showed that it was cirrhotic without gross destruction of the lobular architecture. Amyloid deposits were found mostly at the peripheral areas of the portal tracts.

Case No 266 A L FEMALE AGED 47 Biliary cirrhosis of either the primary or secondary type, extent moderate—In 1953 three and a half years before admission cholecystectomy had been done in a country hospital. Three days later a ligature was removed from around the common hepatic duct. The jaundice persisted and she was re explored twice in that year but no obstruction to the bile passages was found on either occasion. There was however marked cirrhosis of the liver. She had a severe haematemesis early in 1955 and three months before admission a haemorrhage from a stich abscess in the anterior abdominal wall. On examination she was jaundiced and showed some obvious spider naevi on the chest. The abdomen which is illustrated in Fig 85 p 13 (an infra red photograph) showed puckered scars ventral herniae and many dilated collateral veins. The liver extended four finger breadths below the right costal margin and the spleen two finger breadths below the left. A barium swallow did not show any oesophageal varices. Liver function was considerably deranged thymol turbidity 19.5 units serum proteins albumin/globulin 4/4.3.

No operation was done for this patient because there was no proof that the haematemesis had been due to portal hypertension and there were therefore no present indications for operative treatment. The state of the abdominal adhesions would obviously have made any intervention except to the splenic end of the portal tree a matter of considerable difficulty.

Case No 277 M R FEMALE AGED 85 Biliary cirrhosis secondary in type and moderate extent—Said to have had a cholecystectomy done at another hospital twenty nine years ago. She had been reasonably well till three months before admission when she had had attacks of fever and pain which simulated gall stone colic. She had become jaundiced. This fluctuated with the fever. With full medical treatment and antibiotic therapy she improved but as soon as the antibiotic was withdrawn the fever recurred and her health deteriorated. It seemed that she would continue to go downhill unless something was done and since there was a possibility that the cause of her trouble might be a biliary obstruction it was decided that an operation should be done in spite of her great age. She was therefore explored through a Kocher's incision. The common bile duct was found to be grossly distended and contained a large calculus at its lower end. This is shown in the cholangiogram Fig 120. The gallbladder was also found to be present and contained five calculi. The portal venous pressure was 260 mm water and the liver was found to be cirrhotic. The spleen was also enlarged. Choledochotomy and cholecystotomy were done for the removal of calculi. The patient made an excellent recovery and became free from jaundice and fever. An operation for portal hypertension is not contemplated.

Case No 280 R W MALE AGED 36 Advanced cirrhosis following infective hepatitis—Twenty months before admission to St Bartholomew's Hospital vomited a large quantity of blood. Operation elsewhere showed that he had cirrhosis hepatis. Gastric transection. Post operatively the wound broke down partly on account of

ascites. He remained ascitic and was repeatedly tapped. A month before admission further haematemesis and melaena. The ascites increased in quantity. The



FIG 120

Cholangiogram in Case No 277 to show a large stone in the lower end of a dilated common bile duct. The biliary passages within her cirrhotic liver are not dilated.

only cause for his cirrhosis was an attack of infective hepatitis in 1946. He was given intensive medical therapy for three months but his ascites and oesophageal varices remained unchanged. Porta caval anastomosis was done early in 1957. The

portal pressure was dropped from 550 mm to 280 mm of water. The speed of flow in the portal vein was very slow before anastomosis, 7.5 cm per second afterwards. The stoma measured 21 mm in diameter. The turgidity of the portal bed could be seen to deflate at operation and this relief of portal stagnation is reflected in the second photograph shown in Fig. 80b p. 124. (Clot had to be scraped out of the portal vein during the course of the operation.) The man had a brief episode of stupor lasting one day three weeks after operation but then recovered and returned to full work ten weeks after his porta caval anastomosis, much improved in every respect.

Case No. 298 V. N., FEMALE, AGED 53. Advanced cirrhosis hepatis following malnutrition and infective hepatitis when incarcerated in Germany during the war.—Since the war she had been known to have cirrhosis hepatis but recently she had become jaundiced and, in the belief that this was due to extrahepatic biliary obstruction associated with gallstones, a diseased gallbladder had been removed and the common duct been anastomosed to the duodenum. During the previous few months she had had a series of massive haemorrhages from oesophageal varices. Two operations were done, the first a lieno-renal anastomosis to relieve her portal hypertension and the second a re-investigation of her biliary tract to discover if there was indeed an extrahepatic obstruction to account for her jaundice. Investigation shows that she now has biliary cirrhosis and that her bile ducts are patent and working well. The venogram illustrated in Fig. 34 p. 44 was obtained at the first operation before the construction of her anastomosis which was shown by venography at the second operation to be working satisfactorily.

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REFERENCES

- BLAKEMORE A H & FITZPATRICK H F (1951) The surgical management of the post
- sybutamine *Arch Chr Nederland* 1, 253
- (1954) Varices de l'oesophage accompagnant l'hypertension du système porte Vol I p 293 *L'Hypertension portale et le Dumping Syndrome IV^e Congr Gastro Enterolog e* 1954 Paris Masson et Cie
- BOYD D B (1954) Portal hypertension following stricture of the common duct *Lahey Clin Bull* 8, 217
- BROWN W L (1901) Pylephlebitis *St Bart's Hosp Rep* 37, 53
- BROWNE E Z (1940) Variations in origin and course of the hepatic artery and its branches *Surgery* 8, 424
- BLDD G (1845)
- BUSSEY C D (1)
- BUTLER H 1951
- (1952) 71, 688
- (1954) par 7 159
- 29, 427 *Arch Dis Childh*
- CALVERT J LEHMANN H, SILK E & SLACK W K (1954) Prolonged apnoea after suva methonium, a case study of pseudocholinesterase *Lancet* 2 354
- CARTER R N (1947) particular reference
- CHIL
- CHIL *lin Wschr* 42 422
- Philadelphia and
- LONDON Saunders
- CHILD C B HOLSWADE G R McCCLURE R D Jr GORE A L & O'NEILL E A (1952) Pancreaticoduodenectomy with resection of the portal vein in the macaca mulatta monkey and in man *Surg Gynec Obstet* 94 31
- CHILD C G O'SULLIVAN W D PAYNE M A & McCCLURE R D Jr (1951) Portal venography *Radiology* 57, 691
- CLATWORTHY H W (1936) Address at meeting of Association of Paediatric Surgeons London
- COHN R & HERROD C (1952) Some effects upon the liver of complete arterialization *Surgery* 32 214
- COLE W H IRENIUS C & REYNOLDS J T (1955) Strictures of the common bile duct *Trans Amer surg Ass* 73, 217
- CRAFOORD C & FBENCEKER P (1939) New surgical treatment of varicose veins of the oesophagus *Acta otolaryng Stockh* 27, 422
- CRAIG C M Personal communication
- CRILE G Jr (1953) Treatment of oesophageal varices by transoesophageal obliteration *Surg Gynec Obstet* 96 573
- CROSBY R C & COONEY E A (1946) Surgical treatment of ascites *New Engl J Med* 235, 581
- CRUVEILLIER J (1834) *Traité d'anatomie descriptive* Paris Becher 1834 6 2nd ed Paris Labé 1843 5 4th ed Paris Asselin 1862 71
- CULLINAN E B (1952) Spleen and liver diseases Part II Diseases of the liver *British Encyclopaedia of Medical Practice* 2nd ed Vol XI p 480 London Butterworth
- DAS A & BASU A K (1956) Portal hypertension due to extrahepatic obstruction *Brit med J* 1 325
- DE MARTEL M F (1910) Brief summary of the technique of Eck operation *Rev Chir Paris* 42 1181
- DE SOUSA PEREIRA A ADRIÃO M M & RODRIGUES J L (1949) Étude phlébographique du système porte *Portug med* 33 1
- DETERLING R A Jr POWERS S B & BHONSLAY S B (1954) Use of radioactive sodium in the determination of patency of porta caval shunts *Surgical Forum* Vol 5 p 193 Philadelphia and London Saunders 1955
- DIBLE J H (1951) Degeneration necrosis and fibrosis in the liver *Brit med J* 1, 833
- DIBLE J H HUNT W E PUGH V W STEINGOLD L & WOOD J H F (1954) Foetal and neonatal hepatitis and its sequelae *J Path Bact* 67 195
- DIBLE J H McMICHAEL J & SHERLOCK S B V (1943) Pathology of acute hepatitis (aspiration biopsy studies of epidemic arsenotherapy and serum jaundice) *Lancet* 2 402
- DOCK G & WARTHIN A S (1904) A clinical and pathological study of two cases of splenic anaemia with early and late stages of cirrhosis *Amer J med Sci* 127, 24

PORTAL HYPERTENSION

- ✓DOEHRNER G A, RUIZICKA F F ROUSSELOT L M & HOFFMAN G (1956) The portal venous system *Radiology* 66 206
- DOGLIOTTI A M ABEATINI S & CAMPI L (1954) *La radiologia portale In L'Hypertension portale et le Dumping Syndrome vol 1 p 59 II^e Congr Gastro Enterologie 1954 Paris Masson et Cie*
- (1950) The anatomy of the
vol 39, 305
Lancet 1, 530
in Banti's disease *Brit J*
- ✓DU BOULAY G H, GREEN B & HUNT A H (1957) *Portal and Splenic Venography Brit med J* 3, 89
- DUNLAP R W HALLENBECK G A & HANLON D G (1952) Portal hypertension associated with sarcoidosis and with haemochromatosis report of two cases with splenectomy and spleno renal anastomosis *Proc Mayo Clin* 27, 266
- ECK N V (1877) On the ligation of the portal vein *Voyenno med J* 130 1 Translation *Surg Gynec Obstet* 96, 375 1953
- ELIAS H (1952) In *Liver Injury Trans 11th Conf, April 30 May 1 Josiah Macy Jr., Foundation 1953 p 111*
- ELIAS H & PETTY D (1952) Gross anatomy of the blood vessels and ducts within the human liver *Amer J Anat* 90, 59
- ELLIS E E & HOLMAN H (1953) Portal hypertension accompanying cholangitis due to
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ylecholine
in action of succinylecholine in man
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- FALLONER C (1950) The region of the pancreas *Brit J Surg* 37, 334
- FARQUHAR J D STOKES J Jr COLEMAN C M WHITLOCK K Jr BLUEMEL, L W Jr & GAMBESIA J M (1950) Studies on the use of aureomycin in hepatic disease III A
Amer J med Sci 220, 166
& PILLARD H M (1955) Percutaneous spleno
- ENGSTROM P HAPPEL J & PRENDERGAST P (1955) Experimental evaluation of prolonged hypothermia *Arch Surg* 72, 431
- GIBSON J H & RICHARDS R L (1955) Cavernous transformation of the portal vein
J Path Bact 70, 81
- ✓GILES B & TESCHAN P E (1952) The portal circulation time in cirrhosis of the liver following porta caval anastomosis *J Lab clin Med* 30, 537
- GILFILLAN R S (1950) Anatomic study of the portal vein and its main branches *Arch Surg* 61 449
- GRAHAM BRUCE A (1932) Splenectomy and thrombosis *Lancet* 2 1423
- GRAY H K (1951) The hepatic circulation and ascites *Ann R Coll Surg Engl* 8 354
- GVOZDANOVIC V & HAUPTMANN E (1955) Further experience with percutaneous hepportal venography *Acta Radiol Stockh* 43, 177
- GVOZDANOVIC V HAUPTMANN E, NAUMAN E & OBERHOFER, B (1953) Percutaneous splenic venography *Acta Radiol Stockh* 30, 17
- HABIB D V RANDALL H T & SOROFF H S (1953) The management of cirrhosis of the liver and ascites with particular reference to the porta-caval shunt *Surgery* 34 580
- HALLENBECK G A (1955) Portacaval anastomosis rationale indications and technique *Surg Clin N Amer* 35 1099
- ✓HARPER R A K (1955) The clinical application of portal venography in portal hypertension
Amer J Roentgen 73 755
- HELLER KIEL A (1904) Über traumatische Pfortaderthrombose *Verh dtsch path Ges* 7 182
- HELPS E T W & McDONALD D A (1954) Observations on laminar flow in veins
J Physiol 124 631

REFERENCES

1973 11-12

- HIGGINS W H Jr (1947) The oesophageal varix report of 115 cases *Amer J med Sci* 214 436
- HIMSWORTH H P (1947) *The Liver and its Diseases* 2nd ed 1950 Oxford Blackwell
- HUNT A H (1952) The surgical treatment of Banti's syndrome *Brit med J* 1 4
- (1956) *Practice*
- HUNT A H, LAWRENCE K & WHITELEY M (1956) The hornpipe position *Lancet* 1 881
- HUNT A H & WHITTARD R (1954) Thrombosis of the portal vein in cirrhosis hepatitis *Lancet* 1, 281
- HURWITZ A & YENNER R (1952) Pancreatic cyst an unusual cause of hypersplenism *Arch Surg* 63 933
- JAHNKE E J Jr PALMER E D & BRICK I (1954) The Cruveilhier Baumgarten syndrome *Ann Surg* 140, 44
- JAHNKE E J, PALMER E M, SBOROV V M, HUGHES C W & SEELEY S F (1953) An evaluation of the Budd-Chiari syndrome a clinical *Surg Gynec Obstet* 97, 471
- KEO *phagus: its pathologic and clinical*
- KLE *portal vein its relation in Banti's*
- KNOCKER P (1955) Effects of experimental hypothermia on vital organs *Lancet* 2, 837
- LAHEY F H & CYR H P (1951) The dramatic response to splenectomy in a patient with pancytopenia *Lahey Clin Bull* 7, 130
- LARGE A, JOHNSTON G & PRESHAW D E (1952) The portacaval venous shunt *Ann Surg* 135, 22
- LATNER A L (1950) Regime for treatment of severe and acute liver disease *Brit med J* 2, 748
- LAZZARI J H & RACK F J (1951) Hepar lobatum with portal hypertension successfully treated by porta caval anastomosis *Arch Surg* 62, 295
- LEARMONTH J R (1947) The problems of portal hypertension *Ann R Coll Surg Engl* 1, 299
- LEGER L, ALROT G & VAILLE L (1955) Essais de traitement chirurgical des cirrhoses du foie étude critique de la portalisation de l'artere hépatique *Pr méd* 63, 1290
- LEHMANN H (1954) Pseudocholinesterase *St Bart's Hosp J* 53, 111
- LICHTMAN S S (1949) *Diseases of the Liver Gallbladder and Bile Ducts* 2nd ed London Kimpton
- LINTON R R (1949) The surgical treatment of bleeding oesophageal varices by portal systemic venous shunts with a report of 34 cases *Ann intern Med* 31 794
- (1951) The selection of patients for porta caval shunts with a summary of the results in 61 cases *Ann Surg* 134 433
- LINTON R R & ELLIS D M (1956) Emergency and definitive treatment of bleeding oesophageal varices *J Amer med Ass* 166, 1017
- LINTON R M, JONES C M & VOLWILER W (1947) The treatment by splenectomy and spleno renal anastomosis with preservation of the kidney of portal hypertension *Surg Clin N Amer* 27, 1162
- LIPP W F & LIPSITZ M H (1952) The clinical significance of the co existence of peptic ulcer and portal cirrhosis with special reference to the problem of massive haemorrhage *Gastroenterology* 22, 181

PORTAL HYPERTENSION

LLOYD-THOMAS H G L & SHERLOCK S (1952) Testosterone therapy for the pruritus of

33, 1

MCDERMOTT W V WAREHAM J & RIDDELL A G (1956) Bleeding oesophageal varices a study of the cause of the associated hepatic coma *Ann Surg* 144, 318

McFADZEAN A J S & COOK J (1953) Ligation of the splenic and hepatic arteries in portal hypertension *Lancet* 1, 615

✓ MCINDOE A H (1928) Vascular lesions of portal cirrhosis : *Arch Path Lab Med* 5, 23
McMICHAEL J (1932) The portal circulation I The action of adrenalin and pituitary pressor extract *J Physiol* 75, 241

— (1934) The pathology of hepato lienal fibrosis *J Path Bact* 39, 481

MACPHEE I W (1956) Primary biliary cirrhosis *Lancet* 2, 109

MACPHERSON A I S OWEN J A & INNES J (1954) Hepatic function after operations for portal hypertension *Lancet* 2, 356

✓ MACPHERSON A I S OWEN J A & INNES J (1956) Surgical treatment of portal hypertension results in 64 cases *Lancet* 1, 353

MADDEN J L, LORÉ J M, Jr GEROLD F P & RAYM J M (1954) The pathogenesis of ascites and a consideration of its treatment *Surg Gynec Obstet* 99, 385

MAHONEY E B & HOGO L Jr (1950) Congenital stricture of the portal vein *Arch Surg* 61, 713

✓ MANN J D D, ... K A ... (1951) Blood

MAN ..
MAR et le Dumping Syndrome vol I p 327 IV^e Congr de Gastro Enterologie Paris
Masson et Cie

MARKOWITZ J, RAPPAFORT A & SCOTT A C (1949) Prevention of liver necrosis following
... 305
... icæmia complicating antibiotic therapy

G (1949) Sarcoidosis producing portal
eno renal shunt *Ann Surg* 130, 951

of patients

an *J Lab*

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... 34 614

NICOL C S (1952) Abdominal syphilis *Med ill* 6, 394

NICOL C S & TERRY R B (1951) Late syphilis of the liver treated with penicillin *Brit
J vener Dis* 27, 20

✓ NORCROSS J W FELDMAN I D BRADLEY H F & WHITE R M (1951) Liver function an
attempt to correlate structural change with functional abnormality *Ann intern Med*
40, 1110

OSLER W (1944) *The Principles and Practice of Medicine* 16th ed pp 748 749 ed
Christian H A New York Appleton Century

O SULLIVAN W D & PAYNE M A (1956) The emergency porta caval shunt *Surg Gynec
Obstet* 102 668

PARAF A CHALUT J CAROLI J & PORCHER P (1955) Manométrie splénique et splénoporto
graphie dans les affections du système hémapoietique les pyléphlébites, les cirrhoses
du foie *Rev int Hepatol* 5, 617

PARKER R A & SEAL R M E (1955) Cavernous transformation of the portal vein
J Path Bact 70 97

PATEK A J Jr POST J RAYNOFF O D MANKIN H & HILLMAN R W (1948) Dietary
... 138, 8
... sment of portal venous hypertension

PATE Experimental observations on meat
Ann Surg 143 588

REFERENCES

- PHEAR E A, RUEBNER B, SHERLOCK M & SUMMERSKILL W H J (1956) Methionine toxicity in liver disease and its prevention by chlortetracycline *Clin Sci* 15, 93
- PHEMISTER D B & HUMPHREYS E M (1947) Gastro oesophageal resection and total gastrectomy in the treatment of bleeding varicose veins in Banti's syndrome *Ann Surg* 126 397
- (1953) Pfortaderstammes und über
cirrhosis studied by the three
Laennec's cirrhosis of the liver
surgical steps towards solution
gland on 12th December 1956
the colon and rectum *Presi
Medicine Proc R Soc Med*
50, 112 1951
- REDMANN F, BERKER F, AKALIN M & SERIM F (1955) *Ein Verfahren zur Ermittlung
des Zirkulations-Druckes in der Leber in Verbindung mit der Punktionbiopsie dieser
Organs* Istanbul Bilgi Basim ve Yayınevi
- RENNIE J B (1942) Tests of hepatic efficiency *Glasg med J* 20, 125
- RETZIUS (1835) Anastomose entre la veine porte et la veine cave inférieure *Arch gén Méd*
S2 7, 118
- REY
✓ REY age following
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Johns Hopk
Hosp 88, 368
- ROSENSTEIN P (1912) The treatment of cirrhosis of the liver by construction of an Eck
fistula *Arch klin Chir* 98, 1082
- ROUSSELOT L M (1936) The role of congestion (portal hypertension) in so called Banti's
syndrome *J Amer med Ass* 107, 1788
- (1940) The late phase of congestive splenomegaly (Banti's syndrome) with haema-
temesis but without cirrhosis of the liver *Surgery* 8, 34
- (1949) Combined splenectomy and porta caval shunts in portal hypertension *J Amer
med Ass* 140, 282
- ROUSSELOT L M, RUZICKA F F & DOEHNER G A (1953) Portal venography via the
sp J 56, 542
354) Hepatic blood flow studies in
Forum p 372 Clinical Congress
Philadelphia and London Saunders
ct of shunt Address at meeting of
érateurs pour syndrome de Banti
- (1953) Techniques des dérivations porto caves *Sem Hop Paris* 29, 344
- SAPPEY C (1883) Mémoire sur les veines portes accessoires *J Anat Paris* 19, 517
- SATINSKY V P (1948) Porta-caval shunt for portal hypertension a new approach *Rei
Gastroent* 15, 333
- SENGSTAKEN R W & BLAKEMORE A H (1950) Balloon tamponade for the control of
haemorrhage from oesophageal varices *Ann Surg* 131, 781
- SENN A & BLAKEMORE A H (1955) Treatment of oesophageal varices by portal decom-
pression during a period of 9 years experiences and results *Chirurg* 26 217
- SHACKMAN R, GRABER I G & MELROSE D G (1953) Liver blood flow and general
anaesthesia *Clin Sci* 12, 307
- SHERLOCK M (1955) *Diseases of the Liver and Biliary System* Oxford Blackwell
- SHERLOCK M, SUMMERSKILL W H J & DAWSON A M (1956) The treatment and prognosis
of hepatic coma *Lancet* 2 689
- SHERLOCK M, SUMMERSKILL W H J, WHITE L M & PHEAR E A (1954) Portal systemic
encephalopathy neurological complications of liver disease *Lancet* 2, 453
- SHIMONDS J P (1936) Chronic occlusion of the portal vein *Arch Surg* 111 397
- STOCK F E (1952) The surgical treatment of cirrhosis of the liver *Ann R Coll Surg
Engl* 10 187
- SULLENS W E, STEGMANN F & MEYER K A (1949) Surgical considerations in haemorrhage
of the upper part of the gastro intestinal tract *Arch Surg* 59 1244

- SUMMERSKILL W H J DAVIDSON E A SHERLOCK S & STERN R E (1956) The neuro psychiatric syndrome associated with hepatic cirrhosis and an extensive portal collateral circulation *Quart J Med* 25, 245
- TALMA S (1909) Cirrhosis of the liver *Ann Intern Med* 1, 111
- TANI T (1954) The dependence of portal pressure on external pressure *Ann Surg* 140, 632
- TAYLOR F W & EGBERT H L (1951) Portal tension *Surg Gynec Obstet* 92, 64
- TERRY R (1954) White nails in hepatic cirrhosis *Lancet* 1, 757
- THOMPSON W P (1940) The pathogenesis of Banti's disease *Ann intern Med* 14, 255
- TURNBULL H M (1936) In *The Anaemias* by J Vaughan 2nd ed London Humphrey Milford
- VIDAL M (1903) Traitement chirurgical des ascites *Pr méd* 2, 747
- VOLWILER W GRINDLAY J H & BOLLMAN J L (1950) The relation of portal vein pressure to the formation of ascites—an experimental study *Gastroenterology* 14, 40
- VORHAUS L J, SCUDAMORE H H & KARK R M (1950) Measurement of serum cholinesterase activity in the study of diseases of the liver and biliary system *Gastroenterology* 15, 304
- WADE H J & FRAZER E S (1953) Toxicopathic hepatitis due to Fowler's solution: a case treated with dimercaprol *Lancet* 1, 269
- WALKER E M (1952) The pathology and treatment of portal hypertension *Lancet* 1, 729
- (1953) Portal hypertension *Surgical Progress* 1953 p 202 London Butterworth
- (1954) The place of venous shunts in the treatment of portal hypertension *Ann R Coll Surg Engl* 14, 145
- ✓ WALKER R M MIDDLEMISS J H & NATION E M (1953) Portal venography by intra splenic injection *Brit J Surg* 40, 392
- WALSHE J M (1951) Observations on the symptomatology and pathogenesis of hepatic coma *Quart J Med* 20, 421
- (1953) The effect of glutamic acid on the coma of hepatic failure *Lancet* 1, 1076
- (1955) Glutamic acid in hepatic coma
- WELSH J (1950) The supply and regeneration of the rat liver *Brit J Surg* 37, 583
- WELSH J (1952) Benign ulcers of the greater curve associated with ulcerative colitis and cirrhosis of the liver *Brit J Surg* 39, 303
- WELCH C S (1947) A technique for porta caval anastomosis *Surg Gynec Obstet* 85, 492
- (1950) Portal hypertension *New Engl J Med* 243, 598
- WHIPPLE A O (1939) The medico surgical splenopathies *Bull NY Acad Med* 15, 174
- (1945) The problem of portal hypertension in relation to the hepato splenopathies *Ann Surg* 122, 449
- WHITELEY H J (1953) Atheroma of the portal vein *J Path Bact* 66, 563
- WILSON A CALVERT R J & GEOGHEGAN H (1952) Plasma cholinesterase activity in liver disease its value as a diagnostic test of liver function compared with flocculation tests and plasma protein determinations *J clin Invest* 31, 815

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